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Centre for Veterinary Education
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Anaesthesia and Analgesia

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Speaker Profiles

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Dr Zaki graduated from the University of Sydney in 1992. After two years in mixed-practice, she rejoined the Faculty of Veterinary Science in 1995 as a Clinical Registrar in Anaesthesia. Since 1995 she has been teaching, conducting clinical research and providing clinical service within the University Veterinary Teaching Hospital at the University of Sydney. She became a member of the Australian College of Veterinary Scientists, Anaesthesia and Critical Care Chapter in 1997 and completed the Graduate Certificate in Educational Studies (Higher Education) at the University of Sydney in 2005. Dr Zaki is a staunch supporter of the Veterinary Profession and is a frequent invited speaker at Continuing Education seminars, workshops and conferences. Her research and clinical interests are pain management and intravenous anaesthetics. She is currently undertaking a PhD looking at "Understanding the pain of osteoarthritis". In 2008, Dr Zaki was appointed Director of the University Veterinary Teaching Hospital-Sydney.

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Dr Gabrielle Musk graduated from Murdoch University in 1995 and after 6 years in mixed and small animal practices in South Australia and the UK she started a residency in Veterinary Anaesthesia at the Vet School in Glasgow. She is now a European Specialist in Veterinary Anaesthesia working at Murdoch University and is part way through a PhD based on respiratory physiology at the University of Western Australia.
Her particular interests include ventilation techniques, total intravenous anaesthesia, analgesic infusion regimens and undergraduate anaesthesia education.

Program

SATURDAY 16 MAY 2009

9.00 - 11.00 Clinical pharmacology: opioids, ketamine, tramadol, gabapentin
Sanaa Zaki & Gabrielle Musk

The analgesic armamentarium of the clinician is expanding rapidly and to ensure safe and effective use of these drugs a solid understanding of the mode of action and side effects of each drug is essential. The evolution of the drugs, the points at which they interfere with nociception and their clinical application will be covered and suggested dose regimes will be provided. The practical aspects of opioid pharmacology will be presented and examples of how this information can be used clinically will be outlined. The clinical pharmacology of ketamine, tramadol and gabapentin will also be discussed with a focus on the indications and applications of these new analgesic drugs.

11.00 - 11.30 Morning Tea

11.30 - 12.00 Infusion techniques Gabrielle Musk

The infusion of analgesic drugs will achieve a more stable plasma concentration of drug and therefore a more continuous control of pain. Drugs suitable for infusion and the practicalities and advantages of infusions will be covered. The infusion of opioids, ketamine and alpha 2s will provide a basis for the application of these techniques in the perioperative setting.

12.00 - 12.30 Nerve blocks Sanaa Zaki

Local anaesthetic nerve blocks are an under utilised technique in small animal veterinary analgesia, yet they are a very effective way of providing pain relief. An initial discussion of basic local anaesthetic pharmacology will be followed by practical tips and examples of how to perform nerve blocks. The aim is to facilitate immediate adoption of these techniques by attending practitioners.

12.30 - 1.00 Case studies: multimodal analgesia Sanaa Zaki & Gabrielle Musk

1.00 - 2.00 Lunch

2.00 - 3.30 Monitoring techniques & equipment Sanaa Zaki & Gabrielle Musk

To minimise the complications associated with cardiovascular and respiratory system depression during anaesthesia it is essential to work towards the maintenance of oxygen delivery to the tissues. This session will be divided to look firstly at the physiology of oxygen delivery and then examine each factor that contributes to it. The monitoring techniques and equipment commonly used in a clinical setting will be covered to explore both the relevance of what is being measured and the limitations of the equipment. Pulse oximetry, capnography, blood pressure measurement and urine production will be discussed in detail.

3.30 - 4.00 Afternoon Tea

4.00 - 4.30 Artificial ventilation Gabrielle Musk

Basic techniques for mechanical ventilation will be examined comparing volume and pressure cycled ventilation. The indications and side effects will be discussed to provide a practical approach to patients requiring short to medium term mechanical ventilation. The ventilator and monitoring equipment required will also be presented.

4.30 - 5.30 Managing anaesthetic complications Sanaa Zaki

This final session starts with a discussion of the more common complications and emergencies that arise under anaesthesia, such as hypotension and apnoea. A review of current trends in CCPR will follow with emphasis on the early detection and implementation of effective basic resuscitation, since these are key determinants of patient outcome. Participants will have the opportunity to trouble shoot specific complications that arise in their own practices as well as be advised on setting up CCPR protocols and crash carts.

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Chapter 1

Clinical Pharmacology - Opioids

Dr Sanaa Zaki

INTRODUCTION

The use of opioids to treat both acute and chronic pain is well established, and veterinarians have been using opioids to treat pain in animals for many years. There are numerous opioids available each with differing pharmacokinetic and pharmacodynamic properties, yet veterinarians are often making clinical decisions about pain management with limited knowledge of species specific opioid pharmacology. The ability to map and characterise opioid receptors has improved our understanding of how opioids exert their effect and has helped to explain some of the mechanisms behind the many species differences clinicians have observed.

It must be remembered that despite their efficacy and safety, few opioids are actually licensed for veterinary use. It therefore becomes important that clinicians apply an evidenced based approach when using these drugs “off label”; and clinical decisions should be made on the basis of our current knowledge as evidenced by published data.

In addition, the analgesic effectiveness of opioids will be optimised and the undesirable side effects minimised, if veterinarians make informed decisions about which opioid to use and the frequency of dosing interval. This requires a good working knowledge of opioid pharmacology, including how opioid activity differs between species. For example, knowledge of opioid disposition in cats has increased in recent years, and veterinarians can now develop more appropriate dosing regimes, rather than make pain management decisions in cats that are based on extrapolation from other species such as humans and dogs.

WHAT ARE OPIOIDS?

Opioids are any number of naturally occurring or semi-synthetic opium alkaloid derivatives, and other similar compounds which bind to opioid receptors on cell surfaces in the body, and whose opioid effects are blocked by opioid antagonists. Naturally occurring opioids such as morphine are found in opium (an extract of the poppy *Papaver somniferum*). Opioids are referred to as narcotic analgesics because of their ability to induce drowsiness and narcosis (sleep) in addition to their pain relieving effects. Morphine was first isolated from the opium poppy in 1806. In the 1970's opioid binding sites were identified in mammalian brains.

Based on current understanding, there are three classes of opioid receptors; μ, κ and δ. These receptors have also been classified as OP_3 (μ), OP_2 (κ) and OP_1 (δ) based on the order in which the receptors were cloned. For a period, a fourth class of receptor (σ) was thought to exist. However, it is now not considered to fit the criteria of an opioid receptor. Controversy continues about the exact number of opioid receptor classes, with some researchers believing that the ε receptor is another class of opioid receptor, acting as a binding site for the endogenous opioid, β endorphin. It has also been proposed that there are opioid receptor sub types; however, to date this has not been supported by cloning experiments.

Opioid receptors are located throughout the central and peripheral nervous system. Activation of opioid receptors exerts both excitatory (increased activity, vocalisation, manic behaviour) and depressive (analgesia, respiratory depression, bradycardia, sedation) effects. The overall effect varies between individuals and between species.

PHARMACOKINETICS

When an opioid is administered to an animal, three key processes determine the resultant outcome of the opioid/receptor interaction:

a. Selectivity

Opioids can act at μ, κ, δ, or any combination of the three receptor types.

b. Efficacy

Drugs can be pure agonists (inducing a maximal response once bound to the receptor); partial agonists (inducing a sub maximal response once bound to the receptor regardless of the dose administered); or antagonists (inducing no response once bound to the receptor). Some drugs are agonists at one receptor type and antagonists or partial agonists at another receptor type.

c. **Affinity**

Opioids with a high affinity for the receptor (eg buprenorphine) have a long duration of action which is unrelated to the plasma half life of the drug.

This means when deciding on the suitability of any given opioid in the clinical setting, the following questions need to be considered:

- *Which type of receptor does this opioid act on?*
- *What is the intrinsic activity of the opioid once it binds to the receptor?*
- *How strongly does the opioid bind to the receptor?*

Two pharmacokinetic effects of opioids which limit their use in humans are the development of ***'acute tolerance'*** and ***'dependence'.***

Acute tolerance, whereby a higher opioid dose is needed to produce the same pharmacological effect, has been demonstrated in animal studies where high doses of opioids are given to animals not in pain. However, it has not been demonstrated in the clinical setting. It must be remembered that opioids are most commonly administered to animal patients to manage post operative pain and for relatively short periods of time (4-5 days); unlike in human patients where long term use is more common and the phenomenon is well recognised.

Dependence is defined as the need to continue drug administration after prior exposure to the drug, in order to prevent development of a withdrawal syndrome. Again this is not recognised in animals clinically, although it has been studied and demonstrated in laboratory animals. This could also be explained by differences in how we use opioids in the clinical setting. Short-term use (days), minimal dosing and no mechanism for animal patients to self medicate, most likely reduce the likelihood of withdrawal symptoms developing once opioid administration is stopped.

To minimise any negative impact on animal patients as a result of these two pharmacokinetic effects the following strategies should be adopted:

- Assess patient for pain regularly and administer the minimal effect dose
- Monitor and record the use of opioids and note any reduction in effectiveness
- Rotate opioids and/or use alternative analgesics when therapeutic doses are no longer effective.
- Rotate opioids and/or alternate with other classes of analgesics when long term pain management is required (weeks-months)
- Where pure opioids have been administered consistently for greater than 24 hours, their use should be gradually withdrawn over 12 -24 hrs or substituted for a partial opioid prior to complete withdrawal.

Metabolism

Opioids are highly metabolised by the liver, with most opioids undergoing one or more of the following; glucuronidation, demethylation, dealkylation and hydrolysis. For example, 50% of morphine is conjugated with glucuronic acid by the liver. Buprenorphine is also extensively metabolised (metabolite excreted in bile) with only 1% excreted unchanged in urine. Fentanyl on the other hand, although it is metabolised in liver, metabolites are excreted primarily in urine, with some clearance by lung tissue. Metabolic pathways for many opioids differ between species, and are yet to be fully characterised in cats and dogs.

Remifentanil is unique in that it is metabolised by plasma esterases, and does not rely on liver metabolism.

It should be remembered that some opioid metabolites also demonstrate a degree of activity (e.g. morphine-6-glucuronide is an active metabolite of morphine) and may account for species differences in efficacy and length of action with some opioids.

Bioavailability

Opioids are generally well absorbed from the gastrointestinal tract but undergo extensive first-pass metabolism, resulting in poor bioavailability with both oral and rectal administration. For example, the bioavailability of morphine when administered both orally and rectally is approximately 20%.

Transmucosal absorption of Buprenorphine in the cat has been demonstrated to be as effective as intravenous administration, and this route of administration is fast gaining popularity amongst veterinarians. Transmucosal absorption of Buprenorphine compounded in a cellulose based gel has recently been evaluated in the dog. Results from this study suggest this preparation may be suitable for clinical use.

Opioids are generally well absorbed via the intramuscular (IM) and subcutaneous (SC) route, although SC absorption is not consistent with all opioids in all species. For example, there is now evidence suggesting that SC buprenorphine is poorly absorbed in cats. Pethidine is also best given IM because it is difficult to achieve effect plasma levels via the SC route.

PHARMACODYNAMICS

Opioids are unique in that they demonstrate different pharmacological effects depending on whether pain is present or absent.

Desirable effects = Analgesia and Sedation

1. Analgesia

Analgesia is primarily produced by drug agonist activity at μ and κ receptor sites, but most predictably at the μ receptor site, with μ receptor selective agonists being the most effective analgesics.

The analgesic action of opioids is mediated via three mechanisms:

a. Opioids block transmission of noxious stimuli by acting on pre and post synaptic receptors of primary afferent sensory nerves at the level of the spinal cord.

b. Opioids block transmission of noxious stimuli and increase the amount of descending inhibition by acting on higher brain centres

c. Opioids block transmission of noxious stimuli by acting peripherally on opioid receptors generated in inflammatory conditions. Opioid receptors have been identified on nerve endings and inflammatory cells.

2. Sedation

Sedation can be considered a side effect when using opioids primarily as analgesics. However, clinically this side effect is advantageous because opioids are frequently used as premedicants prior to induction of anaesthesia or simply as sedatives for fractious animals in order to facilitate handling and performing diagnostic or therapeutic procedures.

The most effective sedatives are reported to be butorphanol, pethidine and morphine.

Undesirable side effects = CNS excitation, bradycardia, hypotension, respiratory depression, vomiting/nausea, and gastrointestinal stasis

1. Central nervous system excitation

The intravenous use of some opioids can result in temporary overstimulation of the central nervous system (CNS). For example, rapid intravenous administration of morphine may cause a brief period of excitement, especially when used pre-operatively in an animal that is not in significant pain. “Opioid mania” which is reported to occur in cats, is based on a 1925 study by Joel & Arndts where cats that were administered morphine doses of 20mg/kg developed resultant CNS excitement.

Excitement in horses is also well documented, with animals demonstrating increased locomotor function, compulsive eating behaviour and agitation after opioid administration. Butorphanol, for example, can cause box walking and excitement type behaviour in horses, although combined use of sedatives will reduce this effect. Butorphanol has also been reported to induce a stress response in dogs when administered alone.

Despite the potential to cause CNS excitation, use of μ agonists in animals that are in pain will result in calming of the animal.

2. Respiratory depression

Respiratory depression is primarily a μ receptor action, and is mediated by a decrease in the sensitivity of neurones in the medulla to CO_2 and a delay in the response of the respiratory centre. It often manifests as a decrease in respiration rate (RR) rather than tidal volume (V_t), but not always. Partial agonists cause less respiratory depression than pure agonists, and will reach a ceiling effect. Respiratory depression is not as significant a side effect in animals as it is in humans especially where the animal is in pain. The respiratory depression effects may become significant when high doses of pure opioids are repeatedly used or when other CNS depressants been administered.

The panting that is seen with pure opioids such as methadone and fentanyl, is related to alteration of the thermoregulatory centre in the hypothalamus rather than a manifestation of respiratory depression.

3. Cardiovascular effects

Bradycardia, the main cardiovascular side effect, is centrally mediated (vagal) and can be reversed with the administration of atropine. Bradycardia is most marked with the rapid intravenous administration of high doses of pure μ agonists. Some opioids (morphine and pethidine) can cause histamine release when injected intravenously.

Mu agonists such as morphine cause mild hypotension (mediated via opioid receptors in the brain stem, which inhibit sympathetic tone to the heart). Venodilation (an effect of morphine), results in reduced preload, which may be beneficial in cases of congestive heart failure.

4. Gastrointestinal effects

Opioids cause an initial increase in gastrointestinal (GI) motility, with increased non-propulsive rhythmic contractions and an increase in smooth mm tone (including sphincter tone). This is followed by a period of GI stasis. Clinically, GI stasis does not appear to be a significant problem in cats and dogs. However, development of innappetence after 3-4 days of opioid administration (and beyond) may be attributable to GI stasis. Partial agonists such as buprenorphine & butorphanol, have less GI side effects. Pethidine, although a pure opioid, has a spasmolytic action on the GI tract due to its anticholinergic like activity.

Nausea and vomiting, mediated through the CTZ, can occur after the administration of μ agonists. It occurs most frequently with morphine, when used in animals that are 'pain-free'. Butorphanol on the other hand has been shown to have antiemetic activity in both humans and dogs.

OTHER OPIOID ACTIONS

Opioids have an **antitussive action** at μ and κ receptors. There is an inconsistent association between analgesia and antitussive activity. Codeine and butorphanol are the most potent antitussives.

Pruritis is sometimes seen after epidural or intrathecal opioid administration. The itchiness is limited to the caudal dorsum area. It is thought to be due to low doses of opioids enhancing C-fibre nociceptor activity.

Urinary retention can also occur after opioid administration. It is more likely to occur with epidural injection rather than IV or IM, but can occur regardless of the route of administration. The mechanism by which opioids cause urinary retention is still unclear, but it is thought to be both a central effect via inhibition of the spinal reflex, and a direct peripheral effect on smooth muscle. Opioids decrease detrusor muscle tone and the force of contraction but most likely do not increase urinary sphincter tone. They also decrease the sensation of bladder fullness and the urge to void.

Hospitalised animals receiving opioids should be checked regularly for bladder fullness and have their bladder expressed periodically if they are not urinating independently. In particular, patients that have received an epidural should have their bladder expressed or be catheterised to prevent over distension of the urinary bladder in the immediate post operative period.

SPECIFIC OPIOID ACTIONS

1. Morphine

Morphine is a μ selective agonist that is both an excellent analgesic and effective sedative. It reliably induces emesis in dogs (and cats) that are not already in pain. Morphine has an elimination $T_{½}$ of 1-2 hrs (Merrell et al, 1990), however it persists in CSF much longer than it does in plasma, therefore its duration of action is longer than suggested by its plasma $T_{½}$. The $T_{½}$ in cats is considerably longer (~ 3hrs) than dogs because of the reduced ability of the feline liver to metabolise drugs which undergo glucoronidation. This not only results in a longer half life and therefore the need for less frequent dosing (~ every 4-6 hrs) compared to dogs, but also may explain why clinically, morphine appears to be less effective in cats; given that morphine's metabolites are also active.

2. Pethidine

Pethidine is a μ selective agonist with an atropine like structure. As a result it does not cause bradycardia and has minimal gastrointestinal side effects. In fact, its spasmolytic action makes it useful in animals with gastrointestinal disorders, pancreatitis and urolithiasis. Pethidine is less potent than morphine and has a shorter duration of action. The need for frequent dosing makes it a poor choice for post operative pain management. Although, it's rapid onset of action and sedative properties make it useful as a premedicant. Pethidine should not be administered IV as it can cause significant histamine release and resultant hypotension.

3. Methadone

Methadone is a μ selective agonist. Although equipotent to morphine it appears to be a less effective sedative than morphine. However, when combined with a tranquiliser it does provide effective sedation. Unlike morphine intravenous injection does not cause histamine release, and vomiting rarely occurs when administered pre-operatively. Methadone has a long terminal $T_{½}$ in humans (approx 35 hrs) and a variable $T_{1/2}$ in dogs, but a duration of action after a single dose that is comparable with that of morphine (4-6 hours). Care should be taken with repeated dosing or continuous infusions, as accumulation may occur more readily than with morphine.

4. Fentanyl

Fentanyl is a potent μ selective agonist. At therapeutic doses respiratory depression and bradycardia are more marked than morphine and methadone. It has a rapid onset of action (2-5 minutes) and a short duration of action (5-20 minutes) after a single dose. Fentanyl has a large volume of distribution and a high clearance value (with inactive metabolites), but a relatively long elimination $T_{½}$ (3-5 hrs). This is because it deposits in fat and is then slowly released. Because of its short duration of action, fentanyl is often administered as an infusion. Accumulation becomes significant after infusions of > 2 hrs. When used intra-operatively, inhalational requirements are significantly reduced in both cats and dogs.

Hypnorm is a neuroleptanalgesic combination containing fentanyl and fluanisone, commonly used in small mammals. Innovar is another neuroleptanalgesic formulation combining fentanyl and droperidol.

5. Alfentanil

Alfentanil is a potent μ selective agonist. It has a short elimination $T_{½}$ (0.4 – 2hrs), a small volume of distribution and a moderately fast clearance rate. Duration of action is 2-5 minutes and so is best used as a continuous infusion. Pretreatment with atropine and ventilatory support is recommended. Alfentanil has been shown to greatly reduce anaesthetic maintenance requirements.

6. Buprenorphine

Buprenorphine is a μ selective partial agonist. It has a slow onset of action (30-60 minutes for peak effect) regardless of the route of administration. Buprenorphine binds to and dissociates from the μ receptor very slowly. Its high receptor affinity makes reversal and concurrent use of other opioids less effective. Buprenorphine has a long elimination $T_{½}$ (42 hrs in dog), a large volume of distribution and a moderately fast body clearance. Clinically it has a moderate duration of action (4-8hrs). SC administration does not result in fast enough uptake to achieve a sufficient concentration gradient that allows adequate concentrations to reach the effector site. For this reason, IM injection is recommended unless a loading dose has previously been administered.

In the cat, discrepancies in study outcomes looking at the efficacy of buprenorphine as an analgesic compared to other opioids and other classes of analgesics, may in part be explained by the differences seen between the SC and IM routes of administration.

7. Butorphanol

Butorphanol is a μ selective weak antagonist and κ selective agonist. It is an effective sedative with a rapid onset of action, and reduces anaesthetic requirements when used pre-operatively. However, it has limited analgesic effects, with poor efficacy for somatic pain, and analgesia duration of action of only 1-2 hrs. It partially reverses the analgesic and respiratory depressant effects of pure μ agonists.

8. Naloxone

Naloxone is μ, κ and δ selective antagonist. Its duration of action is approximately 60 minutes. Naloxone is a competitive antagonist that reverses all pure opioid effects, by displacing other opioids and binding to opioid receptors without exerting any agonist effect. Interestingly, naloxone is also indicated as an adjunct treatment for hypotension in the management of septic and endotoxic shock.

Clinical applications:

1. Premedication and sedation: opioids are effective sedatives in addition to their analgesic activity

2. Pre-emptive analgesia: pre-emptive activity of opioids has been demonstrated with pethidine in dogs, and in rodents, but the pre-emptive effect is controversial in humans.

3. Intra-operative administration: an anaesthetic sparing effect has been demonstrated for fentanyl, morphine and other opioids in both cats and dogs.

4. Post –operative and non surgical pain: Opioids are a class of analgesics that offer numerous options for managing pain in animals.

Opioids can be used to treat mild, moderate and severe pain states. Duration of action ranges from a few minutes to several hours, depending on which drug is selected. In addition to traditional routes of administration (SC, IM and IV), opioids can be effectively administration via the transdermal (fentanyl), transmucosal (Buprenorphine), oral (morphine and butorphanol), intra-articular (morphine), epidural (morphine, pethidine) and intrathecal routes.

Strategies to optimise effective use of opioids to treat pain

1. Adopt a multi modal approach to pain management that utilises both pharmacological and non-pharmacological strategies.
2. Titrate opioids "to effect" based on the degree of pain the animal is assessed to be experiencing.
3. Consider rotating opioids in cases where continuous use of one opioid is no longer effective.
4. Make use of non traditional routes of administration to increase efficacy and animal compliance.
5. Consider concurrent use of sedatives. This may enhance the analgesic effect of opioids in some animal patients

NOTES:

NOTES:

Chapter 2

Clinical Pharmacology – Ketamine, Tramadol, Gabapentin

Dr Gabrielle Musk

The continuing evolution of veterinary anaesthesia and analgesia is fast. New drugs rapidly become common and popular options for the clinician and a thorough understanding is essential to use them appropriately, and most importantly, safely. Ketamine, Tramadol and Gabapentin will be discussed here.

KETAMINE

Ketamine is an *N*-methyl-D-aspartate (NMDA) receptor antagonist and produces a state of dissociative anaesthesia at higher doses. Through NMDA antagonist it interferes with the development of ‘wind-up’ or sensitization within the central nervous system. ‘Wind up’ refers to frequency dependent potentiation of the responses of spinal neurons to stimulation of unmyelinated afferent nerves. Surgical stimulation can elicit this sensitization and post operatively manifest as hyperalgesia and chronic pain. Ketamine has been used in human medicine for the treatment of traumatic, neuropathic and chronic pain and for peri-operative pain management. It is also known that the analgesic effects of ketamine outlast the anaesthetic effects – most likely through the modification of ‘wind up’.

The pharmacokinetics of ketamine are well reported in the veterinary literature and ketamine is popular as an induction agent, a sedative agent (in combination with alpha 2 agonist, benzodiazepines and opioids) and an analgesic. It can be administered subcutaneously, intramuscularly (though painful) and intravenously. It is also well absorbed across oral mucous membranes.

Ketamine is unique in that it produces sympathetic nervous system stimulation, increasing the levels of circulating adrenaline and noradrenaline. As a result heart rate, cardiac output, blood pressure and myocardial oxygen requirements are all increased. This sympathetic nervous systemic mediated stimulation overrides the direct myocardial depressant effects of ketamine. This may be clinically significant in very sick patients whose sympathetic nervous system is exhausted.

The respiratory effects of ketamine are characterized by a small increase in respiratory rate or apneustic breathing (prolonged inspiratory hold). Laryngeal reflexes are usually relatively well preserved but intubation should be possible. Ketamine also causes bronchodilation so may be useful for patients with asthma.

Anaesthetic doses of ketamine produce a state of dissociative anaesthesia that produces characteristic electroencephalogram changes and may manifest as muscle rigidity and loss of voluntary limbic control.

Ketamine is metabolized to norketamine which is an active metabolite produced in the liver and excreted by the kidneys. Care should be taken in patients with impaired liver or renal function as the duration of action may be significantly prolonged.

The analgesic effects of ketamine can be appreciated at lower doses and can be incorporated into a balanced anaesthetic regime at a number of points. In cats incorporation of ketamine in the premedication (with acetylpromazine or an alpha 2 agonist and an opioid) is particularly useful. The author adds 5-10 mg/kg of ketamine to acetylpromazine (0.03-0.05 mg/kg) and an opioid for IM premedication in unpredictable and stressed cats. The dissociative effects of ketamine are more profound in dogs so it is best reserved for induction of anaesthesia or infusion. Combined with a benzodiazepine, ketamine is a popular and useful induction agent. The 'depth' of anaesthesia observed during a ketamine based protocol is different to that using propofol, alfaxalone or a barbiturate as the muscles are not as well relaxed. Lastly, ketamine can be used for perioperative pain management by bolus or infusion.

The analgesic properties of ketamine can be appreciated at sub-anaesthetic doses which spares the patient of the psychomimetic properties of the drug. In human medicine relatively low doses provide analgesia when used as part of a balanced anaesthetic technique. Extrapolating to veterinary patients this approach has been investigated but when used as the only analgesic drug ketamine does not provide adequate anti-nociception [1]. This study examined the antinociceptive effects of a 0.5 mg/kg IV bolus followed by an infusion of 10 µg/kg/min. This supports the premise that ketamine should be used in conjunction with other analgesic drugs to complement their effects and form a true multimodal analgesic approach.

Conversely a study examining ketamine for peri-operative analgesia in dogs undergoing ovariohysterectomy found that it was a useful analgesic. The assessment of these patients was based upon physiological and behavioural variables and it can't be assumed that ketamine alone provides adequate analgesia [2].

Ketamine is contraindicated in patients with:

1. Raised intra cranial pressure

2. Raised intra ocular pressure

3. Tachycardia or hypertension

4. A history of seizures

TRAMADOL

Tramadol is a synthetic opioid agonist which is an analogue of codeine. It has been used in human medicine since 1977 and has become relatively widely used in veterinary medicine. Its effects are attributable to both the direct opioid effect (μ receptors only) and the inhibition of re-uptake of serotonin and noradrenaline. It is available as tablets, capsules and as a solution for IV or IM injection. Its analgesic potency is one-fifth to one-tenth that of morphine. The metabolites of tramadol have a higher affinity for μ receptors than tramadol itself and are largely responsible for the analgesic affects attributed to the administration of tramadol. It is reported that the metabolites at 200 times more potent at the μ receptor than the parent drug.

Morphine is the gold standard opioid analgesic and is the agent of choice for severe pain in dogs and cats. Its main adverse side effect is respiratory depression although this is not as marked in dogs as it is in humans. Because tramadol is a weak μ receptor agonist it produces significantly less respiratory depression. In people the analgesic effects are attributed in the most part to the inhibition of neuronal noradrenaline reuptake as well as the inhibition of serotonin reuptake. Equipotent doses of tramadol and morphine in humans have the same analgesic effects for mild-to-moderate pain but there is less respiratory depression with tramadol.

A number of studies examine the analgesic efficacy and pharmacokinetic profile of tramadol in dogs, cats, horses and goats. Some authors report equivalent analgesic efficacy to morphine for moderate to severe post operative pain while anecdotal evidence suggests it is a good adjunct to a peri-operative analgesic protocol [3]. Equipotent doses of tramadol (2mg/kg IV) and morphine (0.2mg/kg IV) were administered to dogs scheduled for ovariohysterectomy. The drugs were given after induction of anaesthesia and before the start of surgery. Post-operative pain and sedation scores were comparable for both groups and it was concluded that tramadol was a useful analgesic for dogs undergoing ovariohysterectomy. At these doses there wasn't a difference in respiratory depression between the groups [3].

Epidural administration of tramadol has also been evaluated and compared to IV administration in a group of dogs undergoing tibial plateau leveling osteotomy. The authors speculated the analgesic effects of tramadol are primarily attributable to the µ receptor agonist effects of the M1 metabolite of tramadol and measured the production of this metabolite following epidural (2mg/kg) and IV (2mg/kg) administration. They found that the analgesia provided by tramadol administered into the epidural space or IV was comparable and that the production of the M1 metabolite occurred sooner after IV administration than after epidural administration [4].

Oral tramadol is becoming popular in veterinary practice for management of chronic pain such as arthritis. It can be used along with non-steroidal anti-inflammatories and is considerably cheaper to dose each day. There is little information describing the pharmacokinetic profile of orally administered tramadol in dogs but it is usually administered at 1-2 mg/kg sid or bid or even tid. The side effects are minimal and rare at these doses. If high doses are given (in the order of 10 fold higher) sedation has been observed, along with loss of appetite, respiratory depression and seizures. A sustained release oral formula of tramadol has been investigated in the dog to determine whether extrapolating from human sustained release oral formulations which are popular. It was found that this was not useful in the dog and there was significant variation in absorption and metabolism [5].

Tramadol has also been investigated in cats but more research is required to correlate the clinical effects to the pharmacokinetics. In cats, it has been documented to have a large volume of distribution and relatively slow clearance suggesting that prolonged dosing intervals may be more appropriate [6].

Given the effects of tramadol on serotonin re-uptake it is possible tramadol will interact with medications with similar effects and care should be taken in these patients.

GABAPENTIN

Gabapentin is an anti-epileptic drug that has been used in humans for the treatment of neuropathic pain. It is thought to work at voltage-dependent calcium channels, although its mechanism of action is not entirely understood. There is little in the veterinary literature about its use as an analgesic but in the clinical setting it is proving an interesting option for patients with severe pain and/or neuropathic pain. Neuropathic pain is often difficult to manage as it does not respond to conventional analgesic therapy as well as non-neuropathic pain. It is characterized by allodynia, hyperaesthesia and intermittent spontaneous pain and can be debilitating.

A series of three case reports describing the efficacy of gabapentin for dogs with neuropathic pain describes the use of 14.3 mg/kg (rounded to 100mg) bid PO for months was the only effective analgesic for one dog with spinal nerve root entrapment. The second dog was diagnosed with neuropathic pain secondary to maxillary neuropathy and did not respond to gabapentin (12.5mg/kg bid PO) administered for month [7].

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Chapter 3

Infusion Techniques

Dr Gabrielle Musk

The intravenous infusion of drugs may be at a continuous or variable rate. The aim of any infusion is to achieve a more stable plasma concentration of drug and therefore a more stable effect site concentration and in turn a continuum of clinical effect (analgesia, sedation, anaesthesia etc).

There are a number of techniques for infusion:

1. Continuous rate infusion (CRI)

2. Variable rate infusion (VRI)
    a. Stepped infusion
    b. Infusion altered according to clinical response

3. Target Controlled Infusion (TCI)

Drugs that are ideal for infusion have a particular pharmacokinetic profile: they have a rapid onset of action, rapid equilibration with the effect site, rapid redistribution from the central compartment (vascular space) and are rapidly metabolised. Some drugs are more appropriate than others. A drug with a slow onset of action and slow redistribution and metabolism is not appropriate for infusion (eg thiopentone). These drugs will accumulate over time and the plasma concentration of drug will increase if the infusion rate is not decreased. Drugs with a favourable pharmacokinetic profile include propofol, fentanyl and remifentanil.

Continuous Rate Infusion (CRI)

The continuous or fixed rate infusion of a drug should create a smooth plane of anaesthesia. If the rate of infusion is not adjusted then the drug will accumulate over time. The plasma concentration and effect site concentration will therefore increase as well and side effects associated with an overdose of the particular drug may occur. If the rate of infusion is too slow then there is potential for an inadequate plasma concentration of drug and if the rate of infusion is too fast there is potential for too high a plasma concentration of drug. Clinically this translates to under or over dosing.

With the exception of remifentanil all drugs will accumulate to some degree. For this reason the rate of infusion should be adjusted according to assessment of clinical effect. In practice variable rate infusions are often most appropriate.

Variable Rate Infusion

Varying the rate of infusion of a drug will help prevent inadvertent overdosing. This method of infusion produces a smooth plane of anaesthesia if adequate drug is delivered and as the rate of infusion is adjusted as the duration of infusion increases less total drug is used. These patients will recover sooner but frequent adjustments must be made to ensure the rate matches clinical requirements. In practice this is the most appropriate method of infusion as the clinician can titrate the drug to the required clinical effect.

Stepped infusions have been developed for some drugs in human anaesthesia practice and an example is the Bristol technique for administration of propofol. An initial bolus of 1 mg/kg IV is followed by 10 mg/kg/hr for 10 minutes, then 8 mg/kg/hr for 10 minutes and 6 mg/kg/hr thereafter. Decreasing the rate of administration every 10 minutes helps prevent accumulation of drug, increasing plasma concentration and deepening anaesthesia. A stepped technique is drug specific and species specific. If followed religiously it is inflexible and it is difficult to superimpose change to clinical effect.

Target-controlled Infusion

Target-controlled infusion (TCI) is the most sophisticated and reliable method of infusing drug to achieve a stable plasma concentration. It requires an infusion pump into which the pharmacokinetic profile of a particular drug in a particular species has been programmed. Variable parameters such as age, weight, concentration of the drug and required target plasma concentration are entered. The software will drive the infusion so that the target concentration is achieved in a short period of time (a bolus of drug is given) and maintained. If the clinician increases the target plasma concentration the device will deliver another bolus to achieve the new target and conversely if the clinician decreases the target plasma concentration the device will stop infusing temporarily.

Target-controlled infusion approximate a stable plasma concentration of drug and the rate of infusion will vary according to the set target, the age and weight of the patient and the duration of anaesthesia. It is more economical in terms of total drug used and changes to the target can be made as easily as changes to a vapouriser. Currently the only TCI device available for veterinary anaesthesia practice is for propofol in the dog [1, 2].

EQUIPMENT AND PRACTICALITIES

If an intravenous infusion is indicated then it is important to consider whether or not 2 venous access points are required. If fluid therapy is expected to be complex then it may be worth having 2 venous catheters or a multi-lumen jugular catheter. The volume into which you dilute the drug to be infused will contribute to the total amount of fluid administered to a patient so, especially in smaller animals, this volume should be taken into account when calculating total fluids administered. Some devices may not be accurate at lower infusion rates so it's important to be familiar with the fluid pump or syringe driver you are using. Combining more than one drug in a bag or syringe makes it impossible to titrate drugs individually so consider whether or not it may be more prudent to keep them separate (eg ketamine and opioids).

Springfusor devices deliver a constant rate infusion of drug over a set period of time. They are cheap and lightweight and do not require electricity. Their ease of use is appealing but they will not deliver at a constant rate as temperature will affect their speed. Nor can the clinician alter the rate of infusion unless the concentration of the solution is changed.

To rapidly achieve therapeutic plasma concentrations a bolus of drug may be useful. Some drugs will rapidly equilibrate between the central compartment and the effect site but others may take longer and the administration of a dose to achieve the desired plasma concentration promptly may be necessary. This bolus can be delivered as part of the premedication or induction or given immediately prior to the infusion.

DRUGS

Opioids

Morphine, fentanyl and remifentanil are probably the most popular opioids for infusion in the peri-operative setting. All are pure mu agonists. Morphine has a relatively slow onset and long duration of action while fentanyl and remifentanil have pharmacokinetic profiles more suitable for infusion. Remifentanil has the ideal profile as it has a context sensitive time (CST) of 3 minutes. This means that regardless of the duration of infusion the plasma elimination half life is only 3 minutes. Other drugs will have progressively increasing plasma elimination half lives as the duration of the infusion increases. The advantage of a constant CST is the titratability of the infusion. The disadvantage is that 3 minutes after the infusion is turned off the plasma concentration is inadequate.

This graph demonstrates the concept of context CST. As the duration of infusion increases the time for plasma concentration to decrease by a half increases for fentanyl, alfentanil, sufentanil and remains the same for remifentanil.

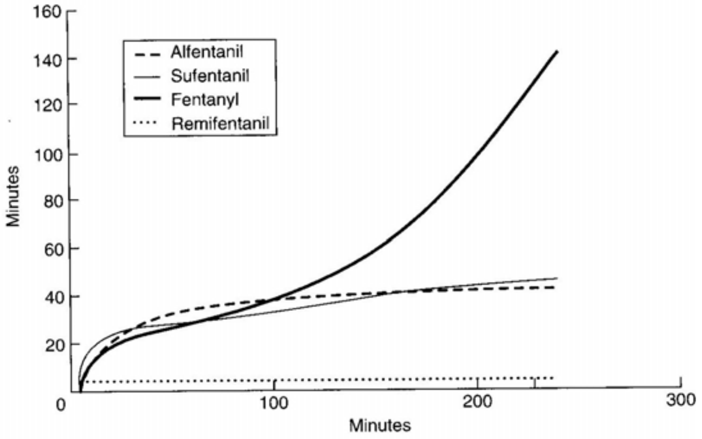

Figure 4.10 Context-sensitive half-live (CSH) of the four opioids.

From ‘Total Intravenous Anaesthesia’. Nicholas L Padfield (2000) Reed Educational and Professional Publishing Ltd U.K. p58.

Indications:

Opioids are indicated for analgesia for mild, moderate and severe pain. They can be used as part of a balanced anaesthetic technique if the delivery of anaesthetic drug must be minimized and are mild sedatives.

Contraindications:

Arguably opioids are never contraindicated but they should be used with caution is a patient is vomiting and morphine is the only opioid available. They should also be used carefully in bradycardic patients or atropine should be readily available. They decrease the heart rate through a vagal mechanism but they do not affect the myocardial directly and have negligible effects on vascular tone. They therefore have negligible effects of blood pressure unless the bradycardia is profound. Often blood pressure improves as there is more time in diastole for ventricular filling.

Side Effects:

The side effects of intravenous opioids are dose dependent and include:

1. Respiratory depression, hypoventilation and hypercapnia
2. Anaesthetic sparing effects so be careful to adjust anaesthetic delivery accordingly
3. Bradycardia
4. Analgesia!
5. Histamine release (especially morphine, though rare)
6. Respiratory stimulation (rare) and panting

Doses:

Morphine: ‘bolus’ 0.1-0.5 mg/kg IM as part of premed or 0.1mg/kg IV prior to infusion at 0.1-0.2 mg/kg/hr

Fentanyl: 0.1-0.7 µg/kg/min

Remifentanil: 0.1-0.7 µg/kg/min

Ketamine

Ketamine is an *N*-methyl-D-aspartate (NMDA) receptor antagonist and produces a state of dissociative anaesthesia at higher doses. It interferes with ‘wind-up’ or sensitization of nociceptors within the central nervous system so is particularly useful for patients with poorly managed or chronic pain. The pharmacokinetics of ketamine during infusion are documented but whether or not a bolus dose is required to achieve an adequate plasma concentration in the first instance is unknown. Presumably a bolus of some sort should be administered and this is easily achieved if ketamine is used as part of the induction of anaesthesia.

Ketamine has been used in cats and dogs for perioperative analgesia and as part of a balanced anaesthetic technique. The doses required to achieve analgesia are lower than those required for anaesthesia. It has been studied in combination with morphine and lidocaine in dogs and with propofol in cats for total intravenous anaesthesia [3, 4]b.

Indications:

Ketamine is indicated for analgesia especially in cases or poorly managed pain (eg road trauma) or chronic pain (eg otitis externa) and surgical cases where direct nerve trauma is likely (eg amputations).

Contraindications:

Ketamine is contraindicated in animals with raised intraocular pressure, raised intracranial pressure and those with a history of seizures. Care should be taken in patients that are tachycardic and hypertensive prior to anaesthesia as the sympathomimetic effects of ketamine may exacerbate either of these conditions.

Side Effects:

The side effects of ketamine by infusion are dose dependent and include:

1. Tachycardia
2. Hypertension
3. Analgesia!
4. Anaesthetic sparing effects
5. Muscle rigidity
6. Increase intraocular and intracranial pressure
7. Dissociation

Doses:

Ketamine: 5-10 µg/kg/min

Medetomidine

Medetomidine is an alpha 2 adrenoreceptor agonist drug that has potent sedative properties. At lower doses it provides useful analgesia and while I reserve it for a plan B approach to analgesia of surgical patients I find it particularly useful in many cases. I am particularly cautious using medetomidine in any patient with cardiovascular disease or altered hydration status but at microdoses the analgesic attributes can be provided without the cardiovascular effects for which alpha 2 agonist drugs create so much paranoia. A good understanding of the biphasic blood pressure response to alpha 2 agonists makes using medetomidine much safer.

Alpha 2 agonists stimulate peripheral alpha 2 receptors causing intense vasoconstriction and hypertension. This vasoconstriction causes a reflex bradycardia and as the drug crosses the blood brain barrier the delayed hypotensive effects occur. Central alpha 2 receptors create hypotension in the face of vasoconstriction and bradycardia. The patient will look vasoconstricted (pale mucous membranes and slow capillary refill time) and may have a profound bradyarrhythmia. These cardiovascular effects can be avoided using much lower doses than the data sheet suggests and for infusion in the perioperative period it is the analgesic effect we are after.

Indications:

Medetomidine is indicated for analgesia when opioids, non-steroidal anti-inflammatories, local anaesthetics and ketamine have failed. It is also useful to achieve cardiovascular stability and sedation in the intensive care unit.

Contraindications:

Medetomidine is never absolutely contraindicated but should be used with extreme caution in patients with cardiovascular disease, hypotension or hypovolaemia. Start at a low dose and titrate accordingly.

Side Effects:

The side effects of intravenous opioids are dose dependent and include:

1. Transient hypertension anaesthetic delivery accordingly

2. Bradycardia and bradyarrhythmias

3. Hypotension

4. Muscle relaxation

5. Sedation

6. Residual vasoconstriction

Doses:

Medetomidine: 0.5-2 µg/kg/hr

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Chapter 4

Local Analgesia Using Nerve Block Techniques

Dr Sanaa Zaki

Local anaesthetics act by blocking neural transmission peripherally and in the spinal cord.

Local anaesthetics are effective analgesics as well as useful adjuncts to general anaesthesia. Veterinarians have been using local anaesthetics such as lignocaine to desensitise tissue in conscious patients through local infiltration techniques for many years. Desensitisation of body regions through the use of nerve blocks has traditionally been reserved for large animals with the added risks of general anaesthesia making standing surgery under local anaesthesia a preferred option.

However, the benefits of using local anaesthetic techniques, in particular nerve blocks and epidurals, in our anaesthetised small animal patients, are now more broadly recognised. Use of local anaesthesia in combination with general anaesthesia can greatly reduce anaesthetic requirements, and therefore anaesthetic risk. It can also facilitate pain management in the immediate recovery period. Nerve blocks applied effectively prior to surgery (pre-emptive) will prevent the transmission of nociceptive information to the spinal cord and potentially reduce the development of central sensitisation.

Local anaesthetics are Na^+ channel blockers. The influx of Na^+ into a nerve cell is required for cell membrane depolarisation and action potential propagation and therefore, impulse conduction. At rest nerve fibres are polarised, with a high extracellular Na^+ ion concentration and a high intracellular K^+ ion concentration. In this rest state ion channels are closed. During depolarisation Na+ channels open to allow an influx of Na+ ions to enter the nerve fibre. Local anaesthetics block these Na channels, preventing depolarisation and therefore reversibly blocking conduction of action potentials. Put simply local anaesthetics stabilise membranes.

The most commonly used local anaesthetics are the amide-linked local anaesthetics, with lignocaine, bupivacaine and mepivacaine being the most common in veterinary practice. These are all metabolised in the liver. They differ in their potency, speed of onset and duration of action. Potency is largely determined by the lipid solubility of the drug. The pKa determines the ratio of ionised to unionised drug and therefore the speed of onset, since only the unionised form can penetrate nerve membranes. Duration of action is a factor of how long the drug stays in contact with the nerve fibre, and so is primarily determined by the degree of protein binding, remembering that highly bound drugs will remain in nerve membrane lipoprotein longer. Blood flow and tissue pH will also influence duration of action.

To prolong duration of action, some local anaesthetic preparations contain a vasoconstrictor (usually adrenaline), so the drug remains at the effector site for longer. These preparations should not be used for epidural injection, injection around an incision site, or for regional intravenous analgesia.

Nerve fibres vary in their susceptibility to local anaesthetic blockade. In the case of myelinated nerve fibres, the larger the nerve fibre the more difficult it is to block. Unmyelinated fibres are more susceptible to blockade because only a small length of nerve fibre membrane requires blockade to prevent transmission of action potentials.

Sensory nerve fibres comprise small, fast conducting, myelinated Aδ fibres (responsible for localised pain eg pin prick) and slower conducting, ,unmyelinated somatic C fibres (responsible for burning/throbbing pain). Motor nerve fibres (Aβ) are large and myelinated, making them more resistant to blockade. This means that pain is usually the first sensation to be blocked, since nociceptor fibres are the most sensitive. Motor blockade follows and requires higher doses. Therefore, where it is undesirable, motor blockade (paralysis) can be minimised by using low effective doses of local anaesthetic.

SYSTEMIC TOXICITY

There are side effects associated with the use of local anaesthetics and these are seen when doses higher than those required for induction of peripheral sensory blockade are used, or where there is inadvertent injection into a vein or artery.

Local anaesthetics have a stimulatory effect on the central nervous system. Animals overdosed with a local anaesthetic will show signs such as restlessness and convulsions, and eventually depression, coma and death.

Local anaesthetics also act on myocardium, decreasing membrane excitability and conduction. Their overall effect is to reduce electrical activity of the heart and decrease the force of contraction. Local anaesthetics can result in decreased BP via their negative inotropic effects as well as a direct vasodilatory action on arterioles.

Lignocaine is used therapeutically to treat cardiac arrhythmias that are of ventricular origin. Use of minimal effective doses reduces the negative inotropic effects. Bupivicaine has the most severe cardiotoxic effects and care needs to be taken not to exceed maximum therapeutic doses and to avoid intravenous injection. Intravenous injection of bupivicaine results in ventricular arrhythmias, significant myocardial depression, and ultimately death.

Cats are considered to be more susceptible to toxicity from use of local anaesthetics. However, in both cats and dogs, toxicity is easily avoided if recommended maximal doses are adhered to (4mg/kg for Lignocaine and 2 mg/kg for bupivacaine) and injection is always preceded by aspiration to ensure the needle is not inside a blood vessel.

LIGNOCAINE

- Available as the injectable hydrochloride salt in stable solutions of 0.5 - 2.0 %
- Topical preparations are available in concentrations of 2% and 4% in viscous and gel forms
- Duration of anaesthesia after infiltration is ~1hr (increasing to 2 hours in the presence of adrenaline)
- Normal therapeutic doses do not directly affect cardiac output, blood pressure or total systemic vascular resistance
- Lignocaine is also used intravenously as an anti-arrhythmic drug to treat ventricular arrhythmia
- Maximum dose for local infiltration and nerve blocks is 4mg/kg.
- It has been shown that the stinging pain sensation of infiltration associated with the use of lignocaine can be greatly reduced by buffering the commercially available preparation.

Combine 9 parts 1% or 2% lignocaine (1.8ml) with 1 part sodium bicarbonate (0.2ml) to make up a buffered lignocaine solution. Buffered lignocaine remains effective for up to 7 days after preparation, so can be made up in advance and refrigerated for storage.

BUPIVICAINE

- Available as 0.25 - 0.75% solutions
- lower onset of action than lignocaine (~ 20 - 30mins)
- 4 times potency of lignocaine
- Duration of action is ~ 2- 6 hrs.
- Toxic if given IV (9 times toxicity of lignocaine)
- Not registered for veterinary use

MEPIVICAINE

- Used widely in equine medicine
- Similar duration of action to lignocaine
- Slightly less toxic than lignocaine
- Useful for lameness diagnosis – causing less post injection oedema

ROPIVACAINE

- Similar onset time and duration of action as bupivacaine
- Intrinsic vasoconstrictor properties so no adrenaline required to prolong action
- Greater safety than bupivicane (less cardiotoxicity)
- Motor and sensory block of shorter duration than Bupivacaine
- Greater sensory blockade selectivity, with motor blockade at higher doses.

OROFACIAL NERVE BLOCKS

1. Mental nerve block

- This nerve block desensitises tissues of the lower jaw, rostral to the 2nd premolar
- The middle mental foramen can be approached either intraorally or percutaneously.
- The foramen is located just caudal to the mandibular labial frenulum in the ventral 3rd of the mandible on the buccal aspect. It sits just ventral to the rostral root of the 2nd premolar in dogs, and at the apex of the mandibular canine tooth in cats. The foramen can be difficult to palpate in small dogs and cats, making this nerve block somewhat problematic.
- If the foramen can't be palpated, use the landmarks to determine needle placement, inject slowly and apply finger pressure over the needle during injection and for 60 seconds after injection to help direct the local anaesthetic into the foramen.
- Injection volume is between 0.1 - 0.2 ml depending on the size of the animal.

2. Mandibular (Inferior alveolar) nerve block

- This nerve block desensitises the bones, teeth and soft tissue (including the tongue) of the lower jaw on the ipsilateral side.
- The mandibular nerve block can be performed using an intraoral or a percutaneous approach.
- For an intraoral approach, the mandibular foramen can be palpated on the lingual surface of the ramus of the mandible, 2/3 of the distance between the last molar and the angular process of the mandible.
- The needle is inserted medially at the level of the root of the last molar and advanced caudally towards the angle of the jaw. The tip of the needle should sit at the entrance to the foramen.
- Injection volume is between 0.2 - 0.5ml depending on the size of the animals, and is intended to block the nerve prior to it entering the foramen.
- For a percutaneous approach, palpate the vascular notch located on the ventral aspect of the mandible just cranial to the angular process.

- Insert the needle at this point along the medial aspect of the bone and advance the needle caudally aiming towards the lateral canthus of the eye, up to the level of the foramen (use your finger to palpate intraorally and confirm needle placement).

3. Infraorbital nerve block

- This nerve block desensitises bone, teeth and soft tissues of the upper jaw, rostral to the 1st maxillary molar
- The infraorbital foramen can be approached either intraorally or percutaneously.
- The foramen can be palpated in dogs, dorsal to the caudal root of the 3rd premolar; and in cats just above the 2nd premolar.
- The needle is inserted in a caudal direction (aiming towards the medial canthus of the eye), parallel and adjacent to the bone. The needle will enter the foramen and at this point correct placement can be confirmed by checking that the needle is well locked in place. Care should be taken in cats and brachycephalic breeds of dog that have a short canal, to ensure the needle is not advanced too far caudally. How far you advance the needle will determine which structures are desensitised.
- Apply digital pressure behind the tip of the needle during injection and for 60 seconds after injection to promote distribution into the foramen.
- Injection volume is between 0.1 – 0.5 ml depending on the size of the animal.

4. Maxillary nerve block

- This nerve block desensitises bone (including the hard palate), teeth and soft tissues (including nose and upper lip) of the ipsilateral upper jaw, rostral to the 2nd maxillary molar
- Palpate a notch on the rostroventral aspect of the zygomatic arch as it meets the bone surrounding the last molar. Insert the needle adjacent this bone, just caudal and medial to the last molar; advancing perpendicular to line of the hard palate. Advance the needle to a depth just beyond the tip of the roots of the last molar.
- Injection volume is between 0.1 – 0.5 ml depending on the size of the animal.

FORELIMB NERVE BLOCKS

1. Brachial Plexus Block

- This nerve block will desensitise all tissues from the elbow to the foot.
- The brachial plexus lies beneath the scapula
- This nerve block is best performed in the anaesthetised patient or under heavy sedation, with the animal lying in lateral recumbency.
- A 22 gauge spinal needle is inserted medial to the greater tubercle (point of the shoulder) and held parallel to the vertebral column while advancing the needle towards the costochondral junction.
- The needle is advanced to the level of the 1st intercostal space and the stylet removed. Aspirate to ensure there is no air or blood, to indicate inadvertent penetration of a blood vessel or the pleural cavity.
- Inject the local anaesthetic while withdrawing the needle approximately 75% of the distance that it was inserted.
- Insert the needle again approximately 30 degrees dorsal with the needle still held parallel to the vertebral column. Repeat the aspiration and injection process.
- Insert the needle again approximately 30 degrees ventral to original direction of insertion, with the needle still held parallel to the vertebral column. Repeat the aspiration and injection process.
- By coursing close to the medial surface of the scapula, damage to underlying tissues structures will be minimised and the two most common complications (inadvertent insertion into the pleural cavity and haemorrhage) will be avoided.

2. Carpal block

- This nerve block targets superficial branches of the radial (dorsal), median (medial) and ulnar (lateral and palmar) nerves.
- It is suitable for dewclaw removal, de-clawing surgery or any injury distal to the carpus.
- The needle is inserted just distal to the carpus, at the proximal-medial aspect of the metacarpus.

- Aspirate and then inject a small volume of local anaesthetic.
- Advance across the dorsum of the foot and insert the needle at the lateral aspect of the metacarpus.
- Aspirate and then inject a small volume of local anaesthetic.
- Locate the small depression just distal to the accessory carpal bone on the ventral aspect of the metacarpus and insert the needle superficially.
- Aspirate and then inject a small volume of local anaesthetic, ensuring infiltration from the medial to the lateral aspect of the foot.

INTERCOSTAL NERVE BLOCK

- This nerve block can be performed immediately following induction of anaesthesia or at the time of surgical closure of the chest wall.
- This nerve block is suitable for animals with rib fractures or those undergoing a lateral thoracotomy.
- There is some overlap of innervation and so it is recommended that 2-3 nerves are blocked cranial and caudal to the affected site.
- The nerves run down the caudo-medial aspect of each rib in association with the ventral branches of the intercostal artery and vein.
- Direct the needle perpendicular to the lateral aspect of the body, and insert onto the rib. The needle is then walked caudally until it penetrates the tissues just caudal to the rib. To optimise the blockade, it should be performed as close as possible to the intervertebral foramen.
- Aspirate before injection the local anaesthetic to ensure the needle has not penetrated the intercostal vein and artery, or entered the pleural cavity.

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Chapter 5

Monitoring the Anaesthetised Patient

Dr Gabrielle Musk

Anaesthesia is the controlled and reversible intoxication of the central nervous where the patient neither perceives nor recalls noxious stimuli. While the delivery of anaesthetic and analgesia agents as part of a balanced anaesthetic approach should achieve both unconsciousness and antinociception it will also depress the cardiovascular and respiratory systems. Direct monitoring of central nervous system depression isn't readily available in a clinical setting so we have to judge the degree of central depression by quantifying things we can measure. We can measure cardiovascular and respiratory system variables which will give us an indication of the degree of central nervous system depression. These combined with the subjective assessments of eye position, palpebral reflexes and jaw tone etc. gives us an idea of 'depth' of anaesthesia.

To ensure normal cellular processes can continue during and after anaesthesia oxygen delivery to the tissues must be maintained. It is especially important that cerebral and myocardial oxygen delivery is sufficient, but other important organs must also receive adequate oxygen to continue functioning.

So, if oxygen delivery is a product of cardiac output and oxygen content of blood:

$$\boxed{\mathbf{DO_2 = CO \times CaO_2}}$$

Where: DO_2 = oxygen delivery

CO = cardiac output

CaO_2 = oxygen content of arterial blood

Therefore, delivery of oxygen to the tissues depends upon adequate perfusion of the tissues AND adequate oxygenation of the blood with which the tissues are being perfused.

And: $\mathbf{CO = HR \times SV}$

$$\mathbf{CaO_2 = (SpO_2 \times 1.34 \times [Hb]) + (0.003 \times PaO_2)}$$

Where: HR = heart rate

SV = stroke volume

SpO_2 = oxyhaemoglobin saturation

[Hb] = haemoglobin concentration

PaO_2 = partial pressure of oxygen in arterial blood

So: $$\mathbf{DO_2 = HR \times SV \times \{(SpO_2 \times 1.34 \times [Hb]) + (0.003 \times PaO_2)\}}$$

To ensure adequate oxygen delivery we therefore need to maintain both cardiac output and oxygen content of arterial blood. From the above equation we can easily measure heart rate, SpO_2 and [Hb] in a clinical setting by auscultating the heart or palpating a peripheral pulse, positioning a pulse oximeter and running a haematology panel. Stroke volume isn’t easily measured without echocardiography and PaO_2 is only measured as part of blood gas analysis. Cardiac output can be measured with sophisticated techniques which are not readily available in a practice environment.

In practice we can be confident that if we adequately monitor the cardiovascular system and respiratory system then each part of the DO_2 equation is accounted for. To break down the equation:

$$\mathbf{DO_2 = \underline{CO} \times CaO_2}$$

Cardiovascular component:

CO may be adequate if heart rate and blood pressure are normal. Arterial blood pressure (ABP) is a product of CO and total peripheral resistance (TPR).

$$ABP = CO \times TPR$$

TPR is not easily measured but some appreciation of vascular tone can be gained from looking at mucous membrane colour and testing capillary refill time. ABP is therefore used as a proxy measure of CO but it should always be remembered that it will be affected by not only CO, but alsoTPR. Normal heart rate will vary between species and breeds but the range within which blood pressure should be maintained is similar between species and breeds.

$$\mathbf{DO_2 = CO \times \underline{CaO_2}}$$

Respiratory component:

CaO_2 should be normal if oxyhaemoglobin saturation is above 95% and the patient is not anaemic. As can be seen from the equation, the major contributor to CaO_2 is the oxygen that is bound to haemoglobin. The oxygen dissolved in the plasma contributes a very small amount to CaO_2. This is because haemoglobin has such a strong affinity for oxygen.

In summary, oxygen delivery to the tissues will be maintained if BOTH perfusion of the tissues is adequate and the blood with which the tissues are being perfused is carrying enough oxygen.

MONITORING EQUIPMENT

Ideally, monitoring should be performed continuously and recorded often (at least every 5 minutes). Anaesthetic record charts are legal documents are must always record as much information about the case as possible. Anything that is monitored should be recorded! How much monitoring is required though? This will vary on a case by case basis but even the most straight forward and routine of cases may surprise so a minimum amount of monitoring should always be applied. As we now know, this should cover both the cardiovascular and respiratory systems so we can be confident that oxygen delivery to the tissues is adequate. In human anaesthesia the question is answered in the general sense that '..continuous monitoring of ventilation and circulation is essential. This may be performed by the use of human senses augmented, where appropriate, by the use of monitoring equipment…'

Minimum standards set by the American Society of Anesthesiologists (ASA) are:

1. Qualified personnel present at all times

2. Continual monitoring of:
    a. Oxygenation
        i. Inspired oxygen concentration
        ii. SpO_2
    b. Ventilation
        i. Observation
        ii. End Tidal CO_2
    c. Circulation
        i. HR, pulse quality, auscultation
        ii. ECG
        iii. Blood pressure
    d. Temperature

Cardiovascular component:

Equipment	Cardiovascular Parameter
Oesophageal stethescope	Heart rate and rhythm, respiratory rate and character
Fingers palpating a peripheral pulse	Pulse rate
Pulse oximeter	Oxyhaemoglobin saturation and pulse rate
Doppler	Systolic blood pressure
DINAMAP or device for non-invasive blood pressure measurement	Systolic, diastolic and mean arterial blood pressure
ECG	Electrical activity of the heart
Urine collection bag connected to urinary catheter	Urine output
Arterial catheter connected to a transducer	Systolic, diastolic and mean arterial blood pressure

Oesophageal stethoscope: this is an inexpensive and useful way to auscultate the heart and lungs during anaesthesia. The end of the blind ending plastic tube is positioned just above the heart and connected to the ear pieces of a stethoscope. It can be left in situ for the duration of the procedure. It does not give any indication of peripheral perfusion or systemic cardiovascular performance. It will only help determine the heart rate, the presence of arrhythmias and abnormal respiratory rate or character.

Fingers palpating a peripheral pulse: the palpation of a peripheral pulse (radial artery, dorsal pedal artery, lingual artery, auricular artery etc) is especially useful to determine the pulse rate and gives a good indication of peripheral perfusion and systemic cardiovascular performance. The quality of the pulse is an indication of the difference between systolic and diastolic blood pressure so does not guarantee a normal blood pressure but it is still a useful qualitative assessment to make. If a peripheral pulse cannot be felt then a more central pulse like the femoral artery should be felt and if this is not easily detected then auscultation of the heart should be performed to determine whether or not the circulation is failing.

Pulse oximeter: this is included in this section of monitoring of the cardiovascular system as it is really just a good monitor of peripheral perfusion and pulse rate. While it gives an oxyhaemoglobin saturation it is a late indicator of respiratory compromise and should not be relied upon as the sole measure of respiratory adequacy. It is placed on a non-pigmented, non-hairy part of the body (tongue, ear, skin fold of the vulva or prepuce, between the toes of white cats etc) and is unreliable if the patient is vasoconstricted.

Doppler: this technique is very useful for monitoring peripheral perfusion, pulse rate and systolic blood pressure. It is the most versatile blood pressure monitor as it can be placed on almost any size patient. A small probe is positioned over a peripheral artery with the concave side down. Some conducting gel is used to ensure a good quality signal. The probe emits an ultrasound beam which is reflected as the blood pulses through the artery, creating a 'schwoozing' sound. The pulse rate can be counted and the systolic blood pressure can be quantified by placed an appropriately sized cuff (cuff width should be approximately 40% of the circumference of the limb) proximal to the probe and inflating the cuff to above systolic blood pressure. This occludes the artery and the sound of the pulsing blood is lost. As the cuff is slowly deflated the 'schwoozing' sound returns. This is systolic blood pressure and should be maintained above approximately 120 mmHg in a dog and 100 mmHg in a cat.

DINAMAP or device for non-invasive blood pressure measurement: DINAMAP = Device for Indirect, Non-invasive, Automated, Mean Arterial Pressure. It employs an oscillometric method of blood pressure measurement where an appropriately sized cuff (cuff width should be approximately 40% of the circumference of the limb) is positioned around a peripheral artery. The machine will regularly inflate the cuff to above systolic blood pressure and then slowly deflate the cuff. Oscillations within the vessel will create oscillations in the air in the inflated cuff which are translated to a systolic blood pressure, when the oscillations commence, mean arterial blood pressure when the oscillations reach maximum intensity, and diastolic blood pressure when the oscillations fade. This equipment is useful and reliable in most settings. It does not perform well if the patient has a bradyarrhythmia, is vasoconstricted or has thick skin or haircoat at the site. It is not particularly useful is horses because their heart rate is so low. It is most accurate for the mean arterial blood pressure and decisions are therefore made on this number. To ensure autoregulation of perfusion to the important organs, mean arterial blood pressure should remain above 60 mmHg.

ECG (electrocardiography): leads positioned to triangulate around the heart will produce a waveform representing the electrical activity of the heart. The ECG will only give information about electrical activity. It does not provide any information on mechanical activity of the heart. While normal electrical activity is essential for co-ordinated myocardial contraction, some form of assessment of mechanical activity must be employed to ensure the heart is actually pumping blood. This can be as simple as palpating a peripheral pulse, positioning a pulse oximeter or measuring blood pressure. The ECG is useful to characterize arrhythmias and gives information about the myocardial environment. It should never be the only monitor of the cardiovascular system. Furthermore, appreciating normal configuration of the ECG is essential to successfully manage any abnormality.

Urine collection bag connected to urinary catheter: if urine output is normal then it can be presumed that renal perfusion is at least adequate. If blood pressure cannot be measured then this technique is useful as the equipment can be fashioned from common clinical apparatus. A urinary catheter is inserted aseptically and connected to an extension set which can then be connected to a collection bag (eg a used fluid bag). Normal urine output is 2 mL/kg/hr.

Arterial catheter connected to a transducer: this is the gold standard method of measuring blood pressure. It is invasive and direct as it requires the placement of a catheter within a peripheral artery. The catheter is connected to extension tubing and a transducer which transmits its signal to a display unit giving a waveform and figures for systolic, diastolic and mean arterial blood pressure. This technique requires skill and practice and the correct equipment and is particularly useful in cases where haemodynamic instability is anticipated, cases with cardiac disease or cases with the potential for severe haemorrhage.

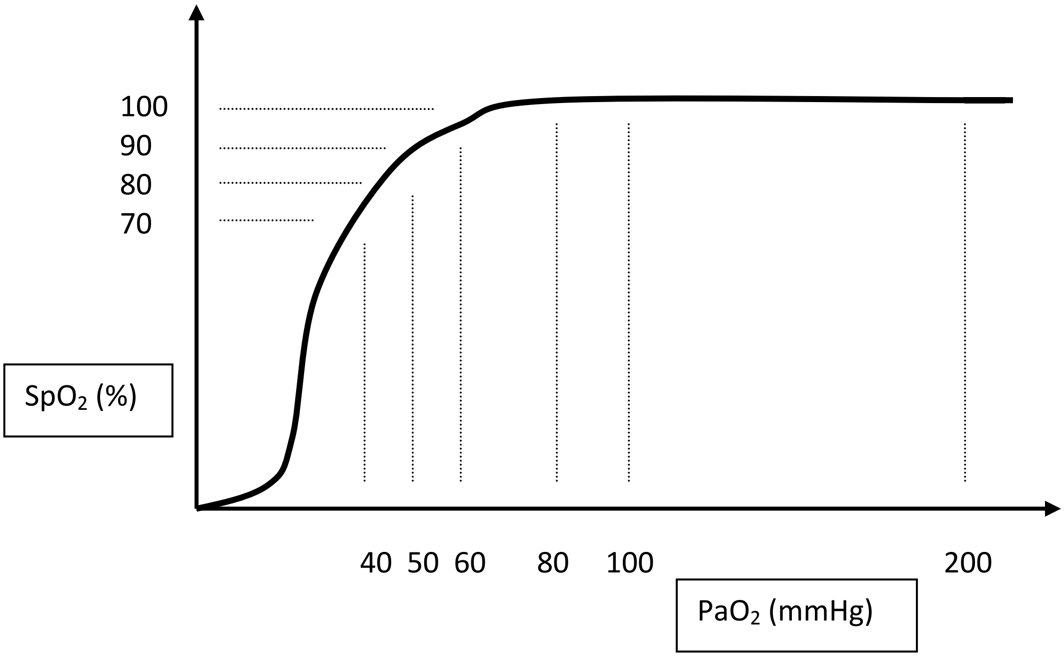
Respiratory component:

Equipment	**Respiratory Parameter**
Observation of chest wall movement	Respiratory rate, effort and character
Oesophageal stethescope	Heart rate and rhythm, respiratory rate and character
Pulse oximeter	Oxyhaemoglobin saturation and pulse rate
Oxygen analyser	Inspired oxygen concentration
Capnograph	End tidal carbon dioxide concentration
Arterial blood gas analysis	Partial pressure of oxygen and carbon dioxide in arterial blood
ApAlert	Respiratory rate

Observation of chest wall movement: this will enable observation of respiratory rate, effort and character. Unusual respiratory patterns can develop under anaesthesia as a direct drug effect (eg apneustic pattern (prolonged inspiratory hold) from ketamine) or as a result of positioning of pathology. In particular, a change in respiratory character should be noted.

Oesophageal stethoscope: this is an inexpensive and useful way to auscultate the heart and lungs during anaesthesia. The end of the blind ending plastic tube is positioned just above the heart and connected to the ear pieces of a stethoscope. It can be left in situ for the duration of the procedure. Listening to the lung sounds will be useful especially if an anaphylactoid reaction or aspiration is suspected.

Pulse oximeter: this is really just a good monitor of peripheral perfusion and pulse rate. While it gives an oxyhaemoglobin saturation it is a late indicator of respiratory compromise and should not be relied upon as the sole measure of respiratory adequacy. It is placed on a non-pigmented, non-hairy part of the body (tongue, ear, skin fold of the vulva or prepuce, between the toes of white cats etc) and is unreliable if the patient is vasoconstricted. Patients are usually on an oxygen enriched gas supply under anaesthesia (anything above 21% oxygen is considered oxygen enriched). Familiarity with the oxyhaemoglobin dissociation curve is important to understanding the limitations of pulse oximetry as a respiratory system monitor.



On the x axis the PaO_2 represents the partial pressure of oxygen dissolved in the plasma of arterial blood. The PaO_2 of a person, a dog, a cat, any mammalian species, is approximately 100 mmHg. This can crudely be calculated as five time the inspired oxygen concentration. So, 21% x 5 = 100 mmHg. If the inspired oxygen concentration is 100% then the PaO_2 should be about 100% x 5 = 500 mmHg. A PaO_2 of 40, 50, 60 mmHg is associated with an oxyhaemoglobin saturation of 70, 80 and 90% respectively. Hypoxaemia is defined as a PaO_2 below 80 mmHg and below this level a small change in PaO_2 causes a big drop in SpO_2. So, you can see that on an oxygen enriched gas supply, the PaO_2 will be well above 100 mmHg so the PaO_2 has to drop significantly, perhaps from 500 to 400 to 300 to 200 to 100 to 80, before any change in SpO_2 is recorded. For this reason the pulse oximeter is not a good monitor of the respiratory system as it cannot give information about early respiratory compromise.

Oxygen analyzer: this is positioned at the common gas outlet of the anaesthetic machine to measure the inspired oxygen concentration. It is essential equipment in human anaesthesia. Most anaesthetic machines will have a low oxygen alarm which will sound if the oxygen supply is interrupted but this device will give you continuous information on the fractional inspired oxygen concentration. This is particularly useful if you are using mixed gases (eg nitrous oxide or air with oxygen).

Capnograph: this is the best measure of adequacy of ventilation and will be discussed by Sanaa Zaki.

Arterial blood gas analysis: while capnography gives a continuous non-invasive measure of the end tidal carbon dioxide concentration arterial blood gas analysis remains the gold standard assessment of acid base status. This includes metabolic and respiratory contributions. The respiratory contribution is primarily determined by the partial pressure of carbon dioxide in arterial blood (normal 35-45 mmHg) and the metabolic contribution by the bicarbonate concentration. These samples need to be analysed soon after collection and stored on ice in the interim.

ApAlert: this is a respiratory rate monitor. It gives limited information on the respiratory system. Minute volume is a product of respiratory rate and tidal volume so the significance of a low or high respiratory rate can only be determined with information about tidal volume. The best way to assess adequacy of ventilation is with a capnograph or serial blood gas analyses. ApAlerts can be misleading and give a false sense of security. I avoid them.

TEMPERATURE

The most common complication associated with general anaesthesia is the development of hypothermia. It occurs as a result of the combination of anaesthetic induced impairment of thermoregulatory control and exposure to a relatively cool ambient temperature and usually vasodilation promoting conductive heat loss. Severe hypothermia can be dangerous and cause myocardial ischaemia, coagulopathies, prolonged healing and increased potential for wound infection. It will also alter the pharmacokinetics of drugs and it is easier to inadvertently overdose a patient. Furthermore, recovery from anaesthesia will be prolonged and shivering during recovery will massively increase oxygen demand in a situation where oxygen delivery may be compromised.

Preventative measures must always be taken, especially in smaller patients. Treatment is difficult and may take an extended period of time. Maintenance of body temperature can be achieved by passively insulating the patient or actively warming the patient with a circulating water blanket, forced warm air blankets, resistive heating (electric blankets), radiant warmers (do not touch the patients) or hot water bottles positioned in areas of high blood flow. Irrigation and intravenous fluids can be warmed and inspired gases should be humidified. Care should always be taken to avoid burns and overheating.

FURTHER READING

‘BSAVA Manual of Small Animal Anaesthesia and Analgesia’ published by the British Small Animal Veterinary Association. Edited by Chris Seymour and Tanya Duke 2007.

Flaherty, D.A. and Musk, G.C. (2005). “Anaesthetic Monitoring Equipment for Small Animals.” In Practice. **27**: 512-521.

Chapter 6

Capnography

Dr Sanaa Zaki

The first infra-red CO_2 measuring and recording apparatus was introduced in 1943. In human anaesthesia capnography has now become a necessary component of standard patient monitoring. Capnography is effective in the early detection of adverse respiratory events; and human morbidity and mortality studies have demonstrated a positive correlation between monitoring with capnography and reduced anaesthetic risk.

However, in veterinary anaesthesia, capnography is still an emerging patient monitoring technique. This is due to the expense of purchasing capnography monitors, and the lack of familiarity with interpreting end-tidal CO_2 readings.

Capnography is a non-invasive method of measuring end-tidal CO_2. End-tidal carbon dioxide is an indirect measure of arterial CO_2 and provides information about tissue metabolism, perfusion and ventilation.

An understanding of CO_2 production and elimination is necessary to enable appropriate interpretation of $ETCO_2$ abnormalities. Capnography not only enables detection of ventilatory and cardiovascular complications, but also equipment failure and technical errors.

HOW DOES CAPNOGRAPHY WORK?

Carbon dioxide is produced by tissues as an end product of cellular metabolism. This is then transported as bicarbonate ions to the lungs for elimination. Capnography samples expired gases during the ventilatory cycle and measures the carbon dioxide content. The end-tidal partial pressure of CO_2 ($EtCO_2$) is a measure of alveolar CO_2 partial pressure and therefore approximates the partial pressure of CO_2 in arterial blood ($PaCO_2$). Interpretation of $EtCO_2$ relies on a number of assumptions being true:

1. CO_2 in capillary blood is in equilibrium with alveolar gas
2. Significant ventilation/perfusion mismatch does not exist
3. The animal's tidal volume is large enough to displace dead space gas
4. Fresh gas flow is not high enough to dilute the sample
5. The sample volume is not high enough to interfere with the animal's ventilation or draw in room air.

Capnography reflects the elimination of CO_2 by the patient to the anaesthetic breathing system. Indirectly it reflects the production of CO_2 by tissues and the transport of that CO_2 to the lungs.

This makes capnography an important non-invasive technique that provides information about CO_2 production, pulmonary perfusion and alveolar ventilation, respiratory patterns as well as elimination of CO_2 from the breathing circuit.

It provides continuous information about airway patency, and is a reliable method for detecting life-threatening conditions arising from technical problems and physiological abnormalities. These include; malposition of the endotracheal tube or inadvertent oesophageal intubation, ventilatory failure, circulatory failure and anaesthetic machine equipment failure. Numerous studies have demonstrated that adverse respiratory events are a major cause of patient injury and death under anaesthesia, highlighting the importance of early detection of such adverse events.

Pulse oximetry is a direct measure of haemoglobin oxygen saturation (SpO_2) and therefore, reflects the patients' oxygenation status and detects the development of hypoxaemia

Capnography on the other hand is an indirect measure that assists with the differential diagnosis of impending hypoxia by providing information about CO_2 production, pulmonary perfusion, alveolar ventilation, respiratory pattern and CO_2 elimination.

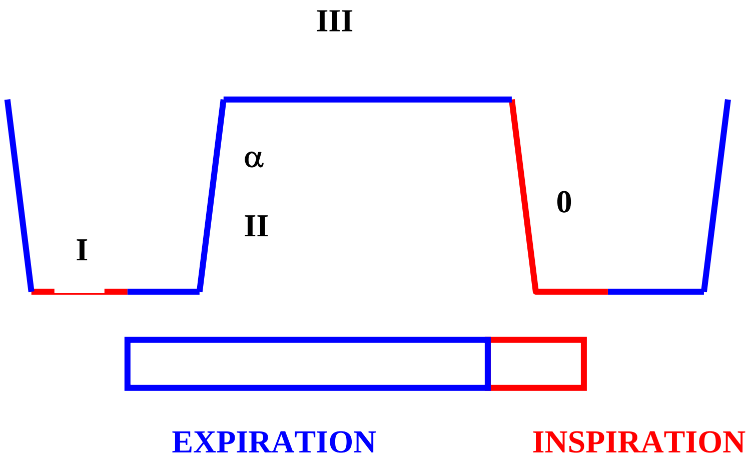
Capnography in combination with pulse oximetry can aid in the early detection of anaesthetic complications and subsequent adverse events that are potentially life threatening. Blood gas analysis is the gold standard for determining arterial O_2 and CO_2 content, by which other methods are compared. Routine use of both Capnography and pulse oximetry to monitor anaesthetised patients minimises the need for repetitive blood gas analysis, while still providing a powerful diagnostic tool.

ANALYSING THE CAPNOGRAM

The CO_2 waveform is consistent in all patients with healthy lungs, and any deviations should be investigated to determine a physiological, pathological or technical cause for the abnormality. When evaluating a capnogram consider the following five (5) characteristics:

- Frequency
- Rhythm
- Height
- Baseline
- Shape

Figure 1: Components of the capnogram



Phase I = dead space (anatomical & apparatus))

Phase II = deadspace (combined anatomical & alveolar)

Phase III = alveolar plateau

α angle = V/Q status of lung

Phase 0 = inspiration

At the beginning of the expiratory phase anatomical dead space gas is exhaled. This should contain no CO_2. The CO_2 content of the exhaled gas rises sharply as more alveolar gas is exhaled, until a plateau is reached representing pure alveolar gas ($EtCO_2$). As inspiration commences, the exhaled gas is replaced with inspired gas, corresponding to a sharp drop in CO_2. This is because there should be no CO_2 in the inspired gas mixture.

TROUBLESHOOTING AN ABNORMAL CAPNOGRAM

This is a simplified list of possibilities that covers some of the more common problems that may be encountered. The capnogram can also be used to detect ventilation/perfusion mismatch.

1. No capnogram:

- Apnoea
- Cardiac arrest
- Disconnection from breathing system
- Extubation
- Esophageal intubation

2. No plateau:

- Intubation problems eg tube is dislodged and within the pharynx or the cuff is leaking

3. Slow rising phase II:

- Partial airway or et tube obstruction

4. Increased EtCO2 (normal baseline):

- Hypoventilation
- Hyperthermia

5. Decreased $EtCO_2$ (normal baseline)

- Hyperventilation
- Cardiac failure (decreased cardiac output; hypotension)
- Shock (e.g. hypovolaemia)

6. Increased $EtCO_2$ (elevated baseline)

- Sticky unidirectional valves

7. Increased or normal $EtCO_2$ with progressively increasing baseline (normal shape curve)

- CO_2 absorber exhausted
- Inadequate fresh gas flow rates

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Chapter 7

Artificial Ventilation

Dr Gabrielle Musk

Spontaneous ventilation is tightly controlled by the information from chemoreceptors feeding to the respiratory muscles via the brain stem. Central chemoreceptors respond to changes in the chemical composition of the blood or other fluid and are situated near the ventral surface of the medulla near the exit of the 9th and 10th cranial nerves. They respond to changes in hydrogen ion concentration and an increase in hydrogen ions will stimulate ventilation and a decrease will inhibit ventilation. Peripheral chemoreceptors are located in the carotid bodies at the bifurcation of the common carotid arteries and the aortic bodies above and below the aortic arch. They respond to changes in the partial pressure of oxygen and carbon dioxide and the pH. The carotid bodies have a very high blood flow and a very fast response rate so even small cyclic changes in blood gases will elicit a response.

The central respiratory centres are in the pons and medulla of the brainstem while voluntary control of breathing originates in the cortex and may override the function of the brainstem to a limited extent. The effectors - the diaphragm, intercostal muscles, abdominal muscles and accessor muscles all work in a coordinated manner to ensure that the partial pressure of carbon dioxide ($PaCO_2$) and oxygen (PaO_2) is maintained within a tight range. Normal $PaCO_2$ is 35-45 mmHg (4.6 – 6 kPa or 4.5 – 5.9%) and PaO_2 should be approximately 100 mmHg when breathing room air. Mechanically ventilated patients should be ventilated to achieve normocapnia (normal $PaCO_2$) and normoxia (normal PaO_2).

Mechanical ventilation replaces or supplements normal ventilation by the pulmonary system. Most of the time controlled ventilation is indicated if carbon dioxide elimination is impaired. Otherwise it is indicated for hypoxaemia, during intrathoracic surgery or for patients that are paralysed. The aim of any ventilation strategy is to recruit, stabilize and maintain open alveoli for efficient gas exchange and matching of ventilation and perfusion.

During normal spontaneous ventilation a negative pressure is created within the thoracic cavity and air is drawn into the lungs. The first mechanical ventilators simulated this negative pressure drawing gas into the lungs and the ‘iron lung’ was one of the first methods of mechanical ventilation. Most modern ventilators force gas into the lungs and create a positive pressure within the thoracic cavity. During positive pressure ventilation, the lungs are inflated by periodically applying positive pressure to the upper airway through a tight fitting mask or through and endotracheal tube or tracheostomy tube. The most efficient ventilation is achieved with the latter two.

During intermittent positive pressure ventilation (IPPV) changes in airway resistance and lung compliance can be overcome by manipulating inspiratory gas flow and pressure. Ventilators will periodically create a pressure gradient between the machine circuit and alveoli that results in inspiratory gas flow. Exhalation is usually passive. The major disadvantages of IPPV are altered ventilation and perfusion relationships, potentially adverse circulatory effects and the risk of pulmonary barotrauma and volutrauma.

Furthermore IPPV will increase physiological dead space because gas flow is preferentially directed to the more compliant, non-dependent areas of the lungs while blood flow favours dependent areas. This may be offset to a degree by the use of positive end expiratory pressure (PEEP).

The haemodynamic effects of IPPV are a result of the positive intrathoracic pressure compromising venous return. As cardiac output is a function of venous return IPPV can drop cardiac output and blood pressure. Care must be taken when the decision to ventilate a hypotensive or hypovolaemic patient as they will be particularly vulnerable to the hypotensive effects of IPPV. Conservative inflation pressures or tidal volumes and a high respiratory rate may be more appropriate in these cases.

BAROTRAUMA

Barotrauma is lung injury resulting from high peak inflation pressures. Diseased lungs are more vulnerable to barotrauma and particular care must be taken when choosing peak inspiratory pressures (PIP). A maximum PIP of 20 cmH_2O is usually ‘safe’ in health lungs. In extreme cases of barotrauma, gas from overdistended, ruptured alveoli form interstitial pulmonary emphysema and tracks along the adventitia to intrapulmonary blood vessel. Eventually the gas bubbles coalesce as they migrate centrally and mediastinal emphysema occurs. If the process persists a pneumothorax may develop.

VOLUTRAUMA

Volutrauma occurs as a result of the repeated opening and closing of normal or disease lung. Shearing forces injure the lung and exudative pulmonary oedema may form as a result of the increased alveolar permeability. Lung compliance will deteriorate and the tidal volume is displaced to more compliant areas and the injury is propagated. High inspired oxygen concentrations will exacerbate the insult.

Ventilator induced lung injury can be minimized if judicious monitoring of ventilated patients is performed and sensible decisions are made about pressures, volumes and inspired oxygen concentration.

VENTILATOR TERMINOLOGY

PIP = peak inspiratory pressure = maximum pressure achieved at the end of inspiration. PIP should not exceed 20-25 cmH_2O in patients with normal healthy lungs.

PEEP = positive end expiratory pressure = the pressure applied by the ventilator at the end of expiration to ‘splint’ the lungs open and prevent alveolar collapse. Increasing the PEEP will help stabilize recruited alveoli but may have adverse haemodynamic effects. PEEP should be set between 1-5 cmH_2O in patients with normal healthy lungs.

I:E ratio = inspiratory time to expiratory time ratio. During spontaneous ventilation this is usually 1:2.5 or 1:3 so it’s best to simulate this. More time is spent in expiration as it is a passive process. Gas trapping is possible if the I:E ratio approaches 1:1.

t_I = I time = inspiratory time. Some ventilators allow you to set the inspiratory time. Altering the t_I will alter the I:E ratio if the respiratory rate stays the same. The longer t_I may be indicated if slower inflation of the alveoli is required. Less time will be spent at the PIP. A short t_I will deliver the breath faster and expand the alveoli more rapidly. This may be more injurious. t_I of 0.5s is common.

V_T = tidal volume. If the tidal volume can be set on the ventilator it is common to set it at 10-15 mL/kg which is considered normal tidal volume for a spontaneously breathing patient. The volume of gas delivered is e a function of PIP especially if compliance is poor. The more compliant the lung, the higher the V_T achieved for a set PIP.

MODES OF VENTILATION

There is a plethora of ventilators available for ventilation during anaesthesia or in critical care setting. Ventilators are usually classified according to the characteristics of inspiratory flow and the factor that terminates inspiration. The types of ventilator most commonly found in veterinary practice are volume-cycled ventilators and pressure-cycled ventilators.

Volume-cycled ventilators terminate inspiration when a preselected volume is delivered. Most of these ventilators will alarm or terminate delivery of the V_T if inspiratory pressure exceeds a pressure limit. A percentage of V_T will be lost to the compliance of the breathing system during inspiration. The higher lung compliance, the more is lost to the equipment. To accurately assess V_T a spirometer must be placed in between the endotracheal tube and the breathing system. Some volume-cycled ventilators do not terminate delivery of the V_T at a particular pressure, they will just alarm so these patients must be watched continuously.

Pressure-cycled ventilators cycle into the expiratory phase when airway pressure reaches a predetermined level. V_T and t_I vary according to airway resistance and lung and equipment compliance. A significant leak in the system will prevent the necessary rise in circuit pressure and therefore cycling between inspiration and expiration. Conversely a sudden increase in circuit pressure (eg kinked tube or obstruction with blood or mucous) will cause rapid cycling between inspiration and expiration and the delivery of smaller V_Ts. Pressure-cycled ventilators are useful for small patients and paediatric patients and are arguably associated with less barotrauma than volume-cycled ventilators if appropriate pressures are set. It is useful to assess V_T during ventilation to appreciate lung compliance.

MONITORING THE VENTILATED PATIENT

A ventilated patient should be continuously observed. Changes in airway and equipment compliance can occur very quickly and must be recognized early to prevent ventilator induced lung injury or ineffectual ventilation. The aim of ventilation is to deliver oxygen and remove carbon dioxide. To achieve this aim it is essential to measure each. Continuous monitoring of oxyhaemoglobin saturation (SpO_2) can be achieved with a pulse oximeter and the fractional inspired oxygen concentration (FiO_2) should be titrated to achieve an SpO_2 between 95% and 100%. The closer the FiO_2 to 21% (equivalent to room air), the better. High FiO_2 for prolonged periods should be avoided to prevent the development of oxygen toxicity.

Continuous monitoring of adequacy of ventilation is possible with a capnograph positioned between the endotracheal tube and the breathing system. This will give breath to breath information on the concentration of carbon dioxide at the end of expiration. In a patient with reasonable ventilation and perfusion matching this will reflect the partial pressure of carbon dioxide in arterial blood and is the best way to assess and alter ventilator settings. Capnography is an invaluable tool for the clinician managing a ventilated patient. Normocapnia is defined as a $PaCO_2$ between 35 and 45 mmHg. Permissive hypercapnia is well tolerated in healthy patients and it maybe useful as if slightly increased the CO_2 will stimulate the sympathetic nervous system and help maintain heart rate and blood pressure. Beyond ‘permissive hypercapnia’ ($PaCO_2$ > 55-60 mmHg) the direct vasodilatory effects of CO_2 may create cerebral vasodilation and an increase in intracranial pressure and it will behave as a narcotic itself. Hypocapnia (due to hyperventilation) may be useful in patients with high intracranial pressure as it causes cerebral vasoconstriction. It may however compromise blood flow to important tissues and is undesirable for prolonged periods of time.

Serial arterial blood gas analysis is also useful to accurately assess oxygenation and ventilation. Normal values are:

pH - 7.35-7.45

$PaCO_2$ – 35-45 mmHg

HCO_3^- - 21-27 mmol/L

PaO_2 – 80-100 mmHg on room air or 5 x FiO_2

FURTHER READING

1. Dugdale, A., *The ins and outs of ventilation 1. Basic principles.* In Pract., 2007. **29**(4): p. 186-193.
2. Dugdale, A., *The ins and outs of ventilation 2. Mechanical ventilators.* In Pract., 2007. **29**(5): p. 272-282.

NOTES:

Chapter 8

Anaesthetic Complications and Emergencies

Dr Sanaa Zaki

Preventing an emergency from occurring is often easier than treating it. However anaesthetic complications and emergencies will occur and the clinician must be prepared for this. The speed with which a problem is detected and corrected correlates directly with the quality of the animal's recovery and its ultimate survival.

Successful resolution of all anaesthetic complications is based around three (3) key elements

1. Early detection

2. Management

3. Evaluation and reassessment of patient

Below is a summary of some of the more common complications that may arise in the anaesthetised patient.

A. EQUIPMENT FAILURE

This may include:

1. The oxygen supply

2. The breathing system

3. Monitoring equipment

When complications arise always consider the possible contribution that equipment failure may be making to the patient's condition.

B. APNOEA/ RESPIRATORY ARREST

Breath holding in response to pungent inhaled agents, the presence of an endotracheal tube, or a sudden noxious stimulus will mimic respiratory arrest due to anaesthetic overdose. Assessing the patient's anaesthetic depth allows easy differentiation between the two.

The period of apnoea is usually brief and does not require any intervention. If it persists beyond 60 seconds or if there is concern that the animal will return to consciousness, it may be necessary to ventilate the animal for a brief period until spontaneous respiration returns. It is important not to over-ventilate the animal at this point so that its respiratory drive is maintained. Respiratory arrest from anaesthetic over dose can arise due to the combined respiratory depressant effects of narcotics, barbiturates, inhalants etc. or due to over all excessive central nervous system (CNS) depression (true anaesthetic overdose).

If apnoea occurs and the animal is deemed to be in a 'deep' plane of anaesthesia, the inspired concentration of the volatile anaesthetic should be lowered or turned off completely, the re-breathing bag evacuated and the patient ventilated with 100% oxygen. The heart sounds and pulse quality should be checked. If the pulse feels weak, then a bolus of intravenous fluids should be administered (5 – 10 ml/kg). If the animal is bradycardic, consider the administration of atropine. The administration of any other drugs that may contribute to CNS or respiratory depression should be terminated eg morphine infusions. If the animal has received any anaesthetic agents that are reversible eg narcotics and $\alpha 2$ agonists, than the antagonist should be administered immediately. Once the animal shows signs of lightening from the anaesthetic the artificial ventilation rate can be decreased to aid in the return of spontaneous breathing. If the vaporiser has been turned off, it can be turned on again at this point, preferably starting on a lower, more conservative setting.

C. AIRWAY OBSTRUCTION

Respiratory obstruction can occur for a number of reasons under anaesthesia. It is important for the clinician to be able to identify an animal that is dyspnoeic due to airway obstruction. Upper airway obstruction commonly occurs under anaesthesia in the brachycephalic breeds and so they should always be intubated. Airway obstruction can occur in any breed, even after an animal has been intubated. An endotracheal tube does not guarantee a patent airway. Remember they too can kink and become blocked with inspissated secretions. Obstructions can also occur within the anaesthetic breathing system, such as when the ‘pop-off’ valve is accidentally left closed. By observing the animal’s chest movements and the re-breathing bag, problems with airway obstruction can be picked up early and corrected. It takes only a few minutes, sometimes less, for an animal to go into cardiac arrest secondary to airway obstruction.

Laryngospasm can occur in any species but cats are most susceptible. It is triggered by irritation of the larynx during light anaesthesia, most commonly at the time of intubation. Although the obstruction is usually partial, animals will become hypoxic and distressed, and so airway patency must be re-established. This is best done by desensitising the larynx with local anaesthetic, deepening the plane of anaesthesia if required, and delivering 100% oxygen to the patient via a mask. The patient may require intubation with a small endotracheal tube. It is most important not to traumatise the larynx during enthusiastic attempts to intubate the animal that is in laryngospasm. Subsequent laryngeal inflammation and oedema will only make things worse. Patients whose hypoxia and airway obstruction persist will require administration of a skeletal muscle relaxant (eg suxamethonium, vecuronium) to enable endotracheal intubation and ventilation.

Bronchospasm, although not seen commonly in small animals, also results in airway obstruction and hypoxia. It is seen more often in cats than dogs, and usually there is a history of airway disease with associated clinical signs. When bronchospasm occurs it is usually severe and life threatening. Artificial ventilation with 100% oxygen will not resolve the hypoxaemia. In fact attempts to ventilate the animal will demonstrate poor lung compliance and an inability to inflate the lungs. Prompt administration of a potent bronchodilator (e.g. isoprenaline or adrenaline) is necessary to open the airways and prevent patient death.

D. HYPOXIA

Hypoxia is defined as a general decrease in oxygen content (ie blood, tissues, and alveoli). **Hypoxaemia** is a decrease in blood oxygen levels.

This is usually defined as a $SpO_2 < 90\%$ or a $PaO_2 < 60$mmHg.

There are four causes of hypoxaemia: (i) hypoventilation;
(ii) diffusion; (iii) shunt; (iv) V/Q mismatch. All may arise under anaesthesia even in animals with normal lungs.

Other causes of tissue hypoxia include: (i) reduced oxygen carrying capacity (eg anaemia); (ii) circulatory deficiency;
(iii) reduction in tissue capability to utilize oxygen (eg cyanide poisoning)

'**Oxygen Flux'** is the total quantity of oxygen delivered to the tissues. It is determined by the total blood flow (cardiac output) and the total arterial oxygen content.

If cardiac output or blood oxygen content is sufficiently low, there will be inadequate tissue oxygenation and tissue hypoxia will develop.

Tissue O_2 delivery = Cardiac Output x blood O_2 content

Cardiac Output

If CO is reduced, more O_2 is extracted from each ml of blood delivered to the tissues to supply their oxygen requirements, and thus venous PO_2 and tissue PO_2 will fall. Most anaesthetic agents cause dose dependent myocardial depression and vasodilation. The overall affect is a reduction in CO.

When CO is greatly reduced the brain and heart are able to maintain reasonable perfusion, however, blood flow to the skin and muscle is reduced. The liver and kidneys have less effective circulatory homeostatic mechanisms than the brain, but cannot withstand hypoxic conditions like the skin and muscle; and so hypoxic damage to these organs may result.

Blood Oxygen Content

Oxygen is carried in the blood in 2 forms:

a) dissolved (~1%)

b) combined with haemoglobin (Hb) (>97%)

Because the majority of O_2 is carried in the HbO_2 form, anaemia will result in reduced blood oxygen content even if the PaO_2 is normal. It is essential to maintain a high cardiac output state when anaesthetising anaemic animals to ensure adequate tissue oxygenation.

Even mild reductions in cardiac output may result in tissue hypoxia. Therefore, it is important to establish and maintain normovolaemia through IV fluid administration and to minimise cardiovascular depression by selecting an appropriate anaesthetic regimen.

Clinical signs of tissue hypoxia include:

1. CNS depression

   This may manifest as an unexplained increase in anaesthetic depth.

2. Cyanosis

   This is the blue discolouration of the skin due to increased levels of deoxygenated Hb in the blood. The quantity of deoxyHb is what determines the degree of cyanosis, not the ratio of HbO2: DeoxyHb.

   Therefore, cyanosis may not be seen if an animal is anaemic or in shock.

3. Changes in the respiratory pattern

   Under deep levels of anaesthesia, a patient may not display an appropriate ventilatory response.

4. Cardiac arrhythmias (primarily VPC's)

Hypoxaemia is most accurately identified by arterial blood gas analysis. However, during anaesthesia, it is most often detected with pulse oximetry, in conjunction with the above signs. Visual observation of mucous membrane colour is not as reliable because cyanosis does not become apparent until the hypoxia is severe, and animals that have pale mucous membranes (common under anaesthesia) will not appear cyanotic even though they may be severely hypoxaemic.

To improve oxygenation of tissues we can:

- If the animal is not already breathing 100% oxygen, increase the partial pressure of oxygen in the alveoli by increasing the inspired O_2 concentration.
- If the animal is hypoventilating increasing minute ventilation by artificial ventilation will also increase the partial pressure of oxygen in the alveoli.

- If the animal is anaemic or there has been significant acute blood loss, increase the O_2 carrying capacity of the blood by administering blood
- If the animal is also showing signs of poor perfusion eg pale mm, hypotension, increase the delivery of oxygen by increasing the cardiac output, that is:
  - decrease anaesthetic depth
  - expand plasma volume
  - use inotropic support
  - contract the vascular space with vasopressors

So when treating hypoxia consider the following:

Is the animal breathing adequately?

Is there a problem with oxygen delivery?

Is there underlying pulmonary disease that could be contributing?

Is cardiac output adequate?

Is the pulse oximeter working properly?

NB: Increased CO is an essential compensation mechanism during anaemia and arterial hypoxaemia. Anaesthesia interferes with this compensatory mechanism.

E. ASPIRATION

This can occur if an animal vomits or regurgitates during induction, maintenance or recovery from anaesthesia. Animals that are at risk of aspiration require special consideration. If an animal vomits or regurgitates one or more of the following may happen:

- Irritation and inflammation of the oesophageal mucosa (oesophagitis)
- Airway obstruction due to the presence of undigested food material
- Aspiration pneumonia
- Acid aspiration syndrome

Of these, airway obstruction and ‘acid aspiration syndrome’ (AAS) pose an immediate anaesthetic emergency. If airway obstruction occurs, the pharynx must be swabbed or suctioned thoroughly. Suction and lavage of the lower airways with saline may also help clear ingesta and dilute its acidity. Position the animal so that gravity is on your side, and consider the use of coupage to clear airways and stimulate the cough reflex. Acid aspiration syndrome results from the inhalation of gastric contents that are acidic (pH < 2.5). The severity of clinical signs is directly related to the volume and pH of the material that is aspirated. The syndrome is characterised by the sudden onset of bronchospasm, tachypnoea, and laboured respiration. Diffuse rales, pulmonary oedema, cyanosis and hypotension follow, with cardiac arrest occurring in severe cases. It is difficult to treat severe cases of AAS and attempts at resuscitation often fail. Treatment involves prevention of further aspiration, suctioning of airways if possible, artificial ventilation with 100% oxygen, and the use of potent bronchodilators such as isoprenaline and adrenaline, and treatment of pulmonary oedema with frusemide. This is definitely an emergency that is easier to prevent than treat!

F. ARRHYTHMIA

Common arrhythmias that may arise under general anaesthesia include sinus bradycardia, 2nd degree atrioventricular block, bigeminy, ventricular premature contractions and ventricular tachycardia.

Bradycardia is defined as a HR below normal, and so the absolute value at which bradycardia is treated depends on the species and size of the animal. Causes of bradycardia include use of opioids and α2 agonists, deep planes of anaesthesia, hypothermia, hypoxia, increased vagal stimulation (e.g. surgical stimulation, pharyngeal manipulation during light planes of anaesthesia).

When bradycardia occurs it is important to assess anaesthetic depth and oxygenation first, rather than simply administering atropine without evaluating the patient.

In addition to treatment of sinus bradycardia, atropine can also be used to treat AV block.

Tachycardia and tachyarrhythmias may indicate a light plane of anaesthesia, excessive surgical (nociceptive) stimulation, hypotension, poor cardiac output, hypercapnoea, hypoxaemia, or hyperthermia. Therefore, it is important to assess the anaesthetic depth and physiological status of the patient prior to attempting management of the tachycardia.

Bigeminy is most often seen in cats under light planes of anaesthesia, and usually does not require the administration of antiarrhythmic drugs. Simply increasing the delivery of anaesthetic to achieve a deeper plane of anaesthesia will resolve the arrhythmia.

A strategy for managing ventricular arrhythmias is set out below:

1. evaluate anaesthetic depth and turn up or down as required

2. treat hypoxia if present

3. treat hypercapnoea if present (IPPV)

4. stop the administration of agents that increase the incidence of catecholamine induced arrythmias (eg if using halothane, switch to isoflurane)

5. assess level of analgesia for the given degree of surgical stimulation, and make sure it is adequate

6. evaluate the effect of the arrhythmia on cardiac output and blood pressure.

7. implement treatment of arrhythmia with lignocaine or other suitable agent, only if it persists and is clinically significant. Ventricular tachycardia and multi focal VPC's should be treated with lignocaine

NB. For ventricular tachycardia and multi focal VPC's treat with Lignocaine first and then follow steps 1-7 prevent recurrence.

G. HYPOTENSION

Hypotension occurs commonly under anaesthesia because inhalation anaesthetics such as isoflurane are potent vasodilators. Mean arterial blood pressure needs to be maintained above 60mmHg to ensure adequate perfusion of vital organs such as the kidneys and brain. This equates to a systolic pressure of approximately 90-100mmHg.

Unfortunately non invasive methods of measuring blood pressure such as the Doppler Ultrasound and the ‘Dinamap’ are least accurate when blood pressure is low. However, low blood pressure readings should not be ignored, especially in already compromised patients. When implementing strategies to manage hypotension consider:

- the patient’s underlying problems
- the anaesthetic depth
- the anaesthetic protocol
- fluid therapy
- vasopressors (drugs that act on blood vessels)
- inotropes (drugs that act on the heart)

The easiest way to improve blood pressure is to reduce the delivery of anaesthetic agent. This may not be possible if the patient is already in a light plane of anaesthesia. Administering a fluid bolus (5-10 ml/kg IV) is another effective way to treat hypotension in some patients. Only if the patient does not respond to fluid administration should pharmacological agents be administered. The exception is animals with primary cardiac disease who may not cope with excessive fluid loading. These animals may benefit from the use of inotropes +/- low dose vasopressors.

H. CIRCULATORY FAILURE

This can be due to failure of the heart, the peripheral circulation or both, and ultimately results in inadequate blood and oxygen delivery to the tissues. Intra operative haemorrhage is an example of this. The blood volume is reduced to a critical level such that the peripheral circulation is no longer adequate even though the heart muscle is still functioning normally. The affected animal goes into ‘shock’.

Cardiac arrest is the ultimate failure of circulation.

This anaesthetic complication must be identified early and treatment commenced immediately for any hope of a positive outcome. A delay of more than 4 minutes from time of arrest may result in irreversible brain damage due to cerebral ischaemia. Absence of an audible heartbeat or palpable pulse are the obvious signs of cardiac arrest, thus diagnosis does not require any elaborate equipment. Under general anaesthesia, where access to the animal may be limited, an oesophageal stethoscope can be an invaluable tool.

Other signs that tell you cardiac arrest is imminent include:

- Dilated pupils that are fixed and not responsive to light
- Dry corneas with no palpebral reflex
- Respiratory arrest or agonal gasping
- A reduction in the amount of bleeding from the surgical site and a dark appearance to the blood
- Ashen mucous membranes (not always cyanotic)

The signs are obvious yes, but only if the clinician takes time to look at the patient.

I. MALIGNANT HYPERTHERMIA

This is an extremely rare condition in dogs and cats but can be life threatening if not recognised. This is a stress or chemically induced genetic myopathy. It has been reported in man, pigs, dogs, cats, horses, birds and wild animals during capture. The pig is the only animal in which it occurs with any regularity and in this species it produces pale soft exudative pork (PSE) syndrome.

The prominent clinical features are:

- rapidly occurring muscle rigour
- explosive and sustained rise in core body temperature (1^0 rise/5-10 mins)
- sinus tachycardia
- tachypnoea
- blotchy cyanosis of skin

Factors that precipitate MH include:

- inhalational anaesthetic agents
- skeletal muscle relaxants
- amide-type local analgesic agents
- ketamine?
- aspirin, alcohol, caffeine
- parasympatholytic and sympathomimetic drugs

The treatment is largely symptomatic. Dantrolene is the only known specific therapeutic drug. It is administered IV 1mg/kg repeated in 5-10 mins. The anaesthetic administration should be stopped and a new uncontaminated anaesthetic machine used to hyperventilate with 100% oxygen. The metabolic acidosis should be corrected with bicarbonate administration, the cardiovascular system supported with IV fluid administration and the animal cooled with ice packs.

RESUSCITATION PROTOCOL FOR ANAESTHETISED PATIENT

1. Stop the administration of any anaesthetic drugs

- Turn the vaporiser off
- Reverse any narcotics etc
- Empty the re-breathing bag and fill with 100% oxygen.

2. Ventilate

- Using 100% oxygen commence artificial ventilation
- Approximately 5-10 breaths per minute depending on the size of the animal.

DO NOT over inflate the lungs or hyperventilate the patient. This will decrease the effectiveness of cardiac massage.

3. Cardiac massage

- Deliver a sharp blow to the centre of the left thoracic wall
- Commence external cardiac compressions. Compress over the heart in cats and small dogs (cardiac pump) using one hand. Compress over the highest point of the chest wall in larger dogs (thoracic pump) using both hands. Aim to compress the chest by 30-50 %, to ensure adequate pressure is achieved to promote ejection of blood from the heart.

Remember the caudal part of the rib cage encases the diaphragm and liver. Avoid it!

- Use as fast a rate as possible that is both effective and sustainable (60 – 160 cpm).

- If the abdominal cavity is already open. It will need to be packed or temporarily closed using a single continuous layer to prevent external displacement of viscera with each compression. Alternatively, internal cardiac compressions can be delivered via a single incision made in the diaphragm. When done correctly, internal massage is more effective than external massage.
- If the thoracic cavity is already open then bypass external massage and deliver internal cardiac massage.

The key to effective CPR is effective cardiac massage. Everything else is secondary until spontaneous cardiac contractility returns.

4. Restore circulating volume

- Following the return of spontaneous cardiac contractions, use a combination of crystalloids and colloids to establish an adequate circulating volume, this is the time when you'll wish you had put a catheter in at induction.
- If surgical haemorrhage was the initiating cause of cardiac arrest. Any source of continued blood loss must be identified and controlled. Successful resuscitation can not occur if this is not achieved, and if circulating volume is not restored.

It is important to gain IV access as soon as possible. However, ***do not stop cardiac compressions to obtain IV access.*** Use any vein which is available and consider using a cut down technique.

5. Pharmacological support

- Table 1 outlines drugs that may be useful during an anaesthetic complication including cardiac arrest and the immediate post resuscitation period, their indications and dose rates.
- Adrenaline, atropine and lignocaine are three drugs you should definitely have on the shelf.

No pharmacological intervention during cardiac arrest has been shown to improve survival rates to hospital discharge.

Adrenaline has potent α1, β1 and β2 effects. It is the "flight or fight" hormone. In a beating heart it increases heart rate and stroke volume resulting in increased cardiac output. It is a potent peripheral vasoconstrictor but causes vasodilatation in muscle beds. It is also a potent bronchodilator of the large and small airways.

It is routinely used in CPR to enhance cerebral and myocardial blood flow by preventing arterial collapse and augmenting aortic diastolic pressure through its alpha effects. However, it has not been shown to improve survival rates in humans. If the decision is made to use adrenaline, it should be administered every **3 – 5 minutes** during the resuscitation because of its short half life.

Atropine competitively blocks the action of acetylcholine on postganglionic parasympathetic cholinergic receptors. It enhances automaticity and conduction in both sinoatrial and atrioventicular nodes. It is useful to treat bradycardia and cardiac arrest where high vagal tone is thought to be a major contributing factor.

Lignocaine is an antiarrhythmic used to treat ventricular tachycardia and VPC's. It does not treat ventricular fibrillation.

6. Defibrillation

- There are basically 4 types of cardiac arrest: ventricular asystole, pulseless ventricular tachycardia, ventricular fibrillation and electromechanical dissociation.
- Ventricular asystole and electromechanical dissociation are the most likely forms of cardiac arrest to respond to cardiac massage and pharmacological intervention. These are also the most common types of arrest that occur in veterinary medicine.
- Ventricular fibrillation is least likely to respond and electro-conversion is often required. This requires an ECG to diagnose and a defibrillator to treat.
- Defibrillator power settings are 5 – 10 Joules/kg for external paddles and 0.5 – 1 joule/kg for internal paddles. Always start at the lower setting and increase gradually if no response.
  The energy discharged is 4-5 joules/kg. If the first attempt is unsuccessful, a second attempt should be made utilising the same level of energy. If this is unsuccessful, basic resuscitation should be continued, adrenaline (1:1000, 1ml/5kg) given and a third attempt made at a higher energy setting.

Adrenaline converts fine fibrillation to course fibrillation which is may be easier to electro-convert to a normal rhythm.

7. Surgery?

- Once the animal is revived and stable, any incisions made during the resuscitation or prior can be closed properly. Lavage any cavities that were opened before closing up. Unless the procedure is 'life saving', surgery that was not completed should be postponed and scheduled for another day.
- After resuscitation the animal may require a low dose of anaesthetic agent or narcotic to immobilise and prevent awareness. Be very careful, you don't want to perform surgery in a conscious animal but you also don't want to overdose a very fragile patient at this point. Consider the use of opioids and muscle relaxants

  Following successful resuscitation for cardiac arrest the animal should be watched carefully for: cardiac arrhythmias, hypotension and/or cerebral oedema and treatment initiated early if any of these occur.

8. Post resuscitation support

Once there is return of spontaneous circulation, it is important to monitor vital organ function and provide support where necessary. The heart, brain and kidneys are most at risk of failure post cardiac arrest and need to be supported by optimising oxygenation and perfusion, and managing complications that may arise secondary to a prolonged ischemic episode.

Animals are susceptible to developing arrhythmias, poor myocardial contractility, seizures, pulmonary oedema, cerebral oedema, renal failure, sepsis, gastrointestinal dysfunction, coagulopathy, and a range of other complex disorders, especially when the period of cardiac arrest has exceeded 5-10 minutes.

A cardiac arrest patient has a high chance of going into cardiac arrest again in the post resuscitation period. The animal should not be left unattended. Continuous observation and monitoring by well trained nursing staff is vital to increase the chance of full recovery. Monitoring instructions need to be specific, with the 'what'. 'when' and 'how' clearly outlined.

What should be monitored?

- mucous membrane colour
- pulse quality
- arterial blood pressure
- heart rhythm (ECG)
- respiratory rate and effort
- level of consciousness and mental alertness
- body temperature
- blood oxygen levels (pulse oximetry &/or blood gas analysis)
- PCV/TPP
- electrolytes, A/B status, blood glucose
- urine production
- capnography (while still intubated)
- central venous pressure?

Post resuscitation support may include some or all of the following:

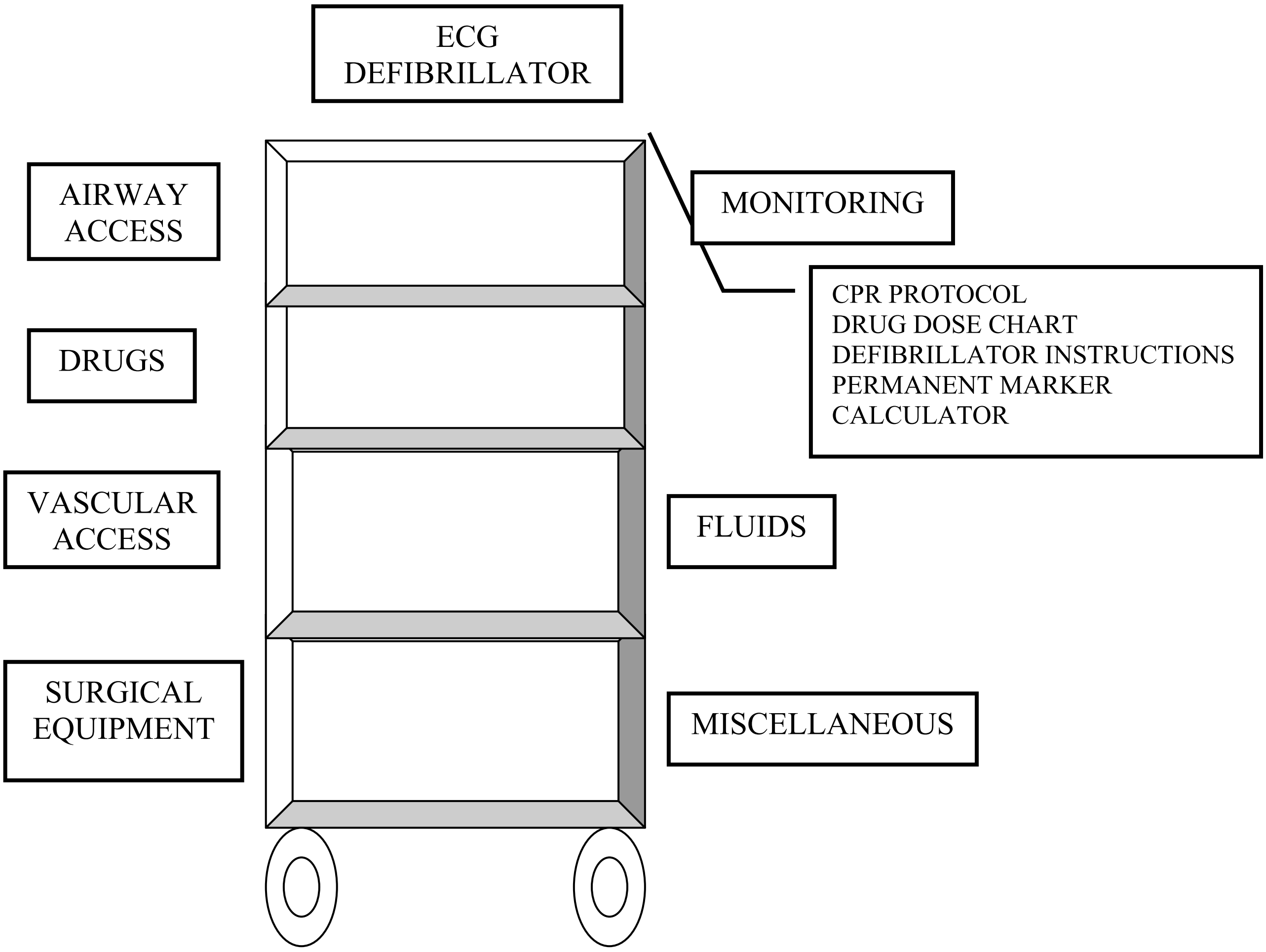
- Artificial ventilation with 100% oxygen until the animal is able to breathe spontaneous and maintain normal CO_2 and oxygen levels.
- Intravenous fluid administration to re-establish an adequate circulating volume and replace losses. Consider use of synthetic colloids as well blood products if significant blood loss has occurred.
- Ongoing use of inotropes and vasopressors to assist in maintaining cardiac output and improve vital organ perfusion. Use of drugs such as dopamine and dobutamine can be titrated to affect based on the animal's response. Mucous membrane colour (in the absence of anaemia), pulse quality, blood pressure, and urine production are all good indicators of perfusion
- Use of diuretics such as mannitol to treat cerebral oedema. This should be considered if the cardiac arrest was prolonged or where spontaneous ventilation and consciousness has not returned.

- Use of diuretics such as frusemide to treat pulmonary oedema. This should be implemented based on clinical signs, chest auscultation +/- blood gas analysis
- Protection of the gastrointestinal tract through the use of mucosal protectants and broad spectrum antibiotics.
- Active warming if the patient is hypothermic.
- Administration of analgesics such as opioids should be commenced once hemodynamic stability has been established.

SETTING UP A CRASH CART

A ‘crash cart’ is simply the dramatic title given to a mobile emergency kit. The key words are ‘mobile’ and ‘emergency’. A crash cart needs to be mobile so that valuable time is not wasted moving an emergency patient from one part of the hospital to another. A crash cart should only contain those items that could be used in an emergency or resuscitation situation. There should be nothing unfamiliar inside the crash cart. Novel drugs and surgical instruments that staff members do not know how to use are useless in an emergency situation. They create clutter and make it difficult to access the items that are needed and will be used in an emergency.

Items need to be clearly labelled and laid out in a logical order, so they are easily accessible. For example, a mobile trolley with several draws makes an ideal crash cart. It can house equipment and drugs in clearly labelled sections, in the order that they may be needed or according to which organ system they are required for, or according to the type of emergency they may be used for. The top of the trolley can then be used for monitoring equipment or a defibrillator. An example of a ‘floor’ plan is given below.



The crash cart needs to be checked regularly to restock used items and dispose of perishable items that have reached their expiry date. The contents of a crash cart will vary from practice to practice depending on the type of emergencies that are encountered and the personal preference of the veterinarians in the practice. Especially with regards to what drugs and fluids are stocked.

Must have items include:

- A resuscitation protocol flow chart
- A calculator
- A drug dose sheet
- A permanent marker
- Pen and monitoring sheets

STRATEGIES FOR SUCCESS

CPR is most successful when cardiac arrest is detected immediately

CPR is most successful when undertaken by a team of staff that are well trained and coordinated

There should always be a **team leader** who directs actions and makes decisions

- A person to ventilate
- A person to deliver cardiac massage (may need to rotate)
- A person to achieve IV access, administer drugs and fluids and set up monitoring
- Someone to record the events

CPR is most successful when the A B C is done well (especially C)

CPR is most successful when the patient has treatable disease

This is the reason why most CPR is not successful

ESSENTIAL VETERINARY SKILLS

- Leadership and clear thinking
- Communication at all levels
- Trouble shooting under stress
- Effective cardiac massage
- A good working knowledge of the crash cart
- A good understanding of cardiovascular and respiratory physiology
- Accurate record keeping
- Basic ECG Interpretation

- Humility and compassion
  (Remember <15% of human in-hospital cardiac arrests survive to discharge)

Table 1: Emergency Drugs Chart: Dose Rates & Indications

Drug	**Dose**	**Indications and uses.**
Adrenaline	Dilute 1:1000 ampoule in 10ml. Give **1ml/5kg**. Or Add 1 – 2 ampoules to ½L 5% Dextrose and titrate to effect. (1 – 10 mg/kg/min)	Cardiac arrest: Potent vasoconstrictor and inotrope. Can convert fine fibrillation to course fibrillation, which is easier to electro-convert. Beware! May trigger fibrillation in Halothane anaesthetised animals that are in asystole. *Also used for Anaphylactic and anaphylactoid reactions.*
Antibiotics	Cephalothin @ 20mg/kg Ampicillin @ 20mg/kg Gentamicin @ 2mg/kg	Sepsis, reflux and aspiration, surgical prophylaxis.
Atropine	0.04mg/kg	Cardiac arrest, vagally mediated bradycardia.
Bicarbonate	1mmol/kg $NaHCO_3$ slowly over 15minutes.	Acidosis eg after an ischaemic episode or cardiac arrest
Calcium	½ - 1 ml/10kg 10%CaCL **Or** ½ -1ml/kg of 10% Calcium Gluconate slowly	Counteract cardiac effects of hyperkalaemia. Effects only last ~ 15 minutes.
Corticosteroids	Methylprednisolone @ 5 – 30 mg/kg Prednisolone @ 5 – 10 mg/kg Hydrocortisone @ 30 – 50 mg/kg	Shock, head and spinal trauma, Addisonian crisis (low end dose rate), post surgical inflammation/oedema of upper airways.
Dopamine	2 – 15µg/kg/min CRI	Dopaminergic effect @ < 5mg/kg/min b effect @ 5 – 10 mg/kg/min a effect @ 10 – 20 mg/kg/min
Dobutamine	1 – 20 µg/kg/min CRI	Ionotropic support eg post resuscitation, hypotension. Beware of tachyarrhythmias @ > 10mg/kg/min
Frusemide	1 – 4 mg/kg	Pulmonary oedema. Oliguric renal failure (to promote diuresis in conjunction with fluid therapy). Low dose for cats.
Glucose	0.5 - 1ml/kg 50% Dextrose 0.25 – 0.4ml/kg/hr 50% Glucose CRI	Hypoglycaemia eg sepsis, insulinoma
Isoprenaline	2 – 10 µg/kg 0.01–0.1µg/kg/min CRI	Bradycardia (not responsive to atropine) Bronchospasm
Lignocaine	0.5 – 1 mg/kg (cat) 1 – 4 mg/kg (dog) 25 – 80 mg/kg/min CRI	Ventricular arrhythmias
Mannitol	0.5 – 1 g/kg slowly over 20 minutes	Cerebral oedema, increased ICP Oliguric renal failure (to promote diuresis in conjunction with fluid therapy).
Nor adrenaline	0.2 – 0.5mg/kg/min ½ ml in 100ml, and give 1drop/sec/20kg	Hypotension
Potassium	0.5 mmol/kg/hr (maximum dose)	Hypokalaemia

# Drugs should be administered intravenously unless alternative route is stated.

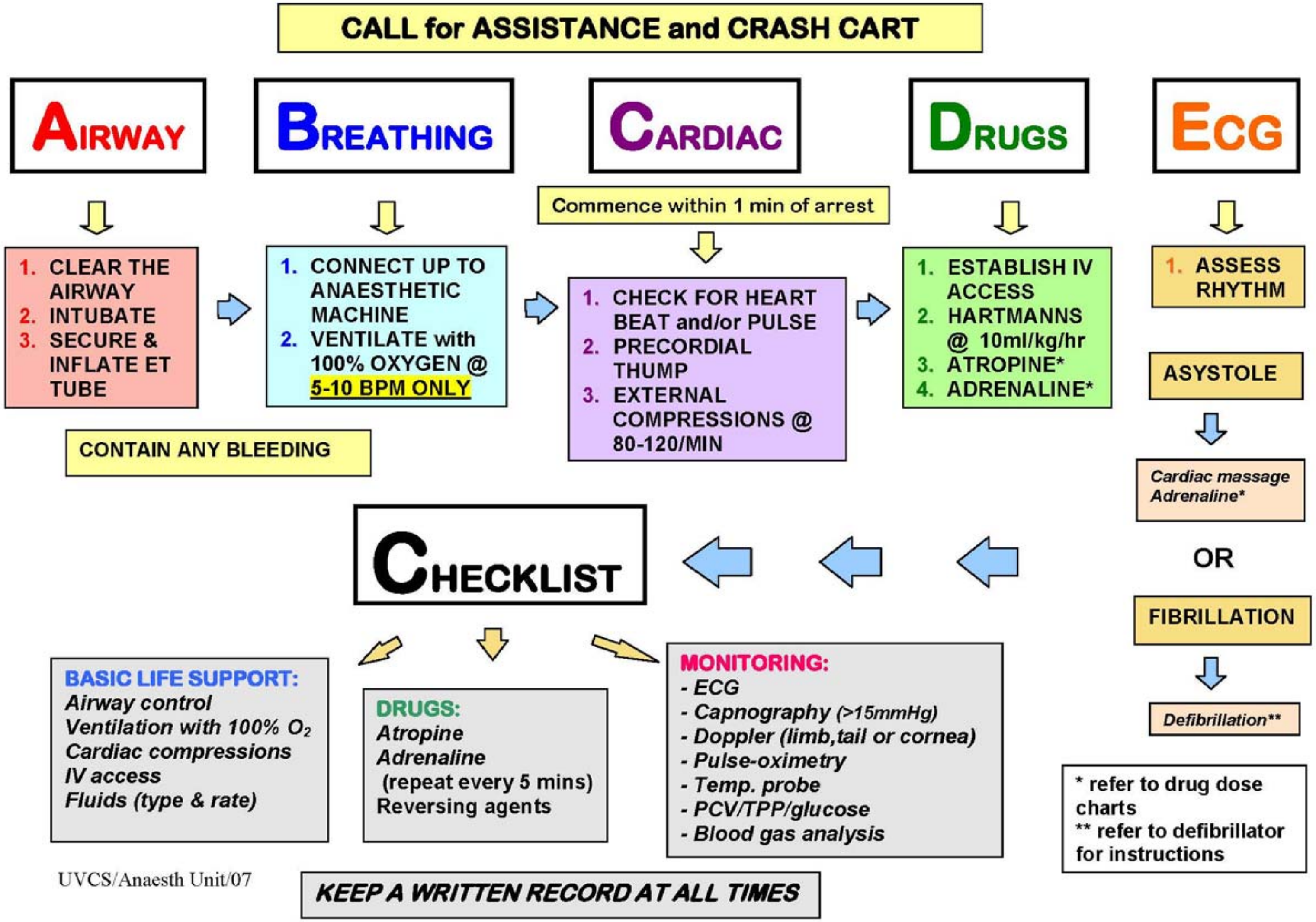
## Doses are for cats and dogs, unless otherwise stated.

CPR DRUG DOSE CHART

	ADRENALINE dog/cat	ATROPINE dog/cat	LIGNOCAINE dog		LIGNOCAINE cat		GLUCOSE dog/cat
DOSE RATE	1ml/5kg	0.04 mg/kg	1 - 4 mg/kg		0.5 - 1mg/kg		0.25 - 0.5g/kg
CONCENTRATION	1:10 000	0.06 mg/ml	20mg/ml		20mg/ml		50% (0.5g/ml)
			MINIMUM	*MAXIMUM*	*MINIMUM*	*MAXIMUM*	
PATIENT WEIGHT (KG)							
2	0.4ml	0.15ml	0.1ml	0.4ml	0.05ml	0.1ml	1ml
5	1ml	0.4ml	0.25ml	1ml	0.13ml	0.25ml	2.5ml
10	2ml	0.7ml	0.5ml	2ml	0.25ml	0.5ml	5ml
15	3ml	1ml	0.75ml	3ml			7.5ml
20	4ml	1.3ml	1ml	4ml			10ml
25	5ml	1.6ml	1.25ml	5ml			12.5ml
30	6ml	2ml	1.5ml	6ml			15ml
35	7ml	2.4ml	1.75ml	7ml			17.5ml
40	8ml	2.7ml	2ml	8ml			20ml
45	9ml	3ml	2.25ml	9ml			22.5ml
50	10ml	3.3ml	2.5ml	10ml			25ml

UVCS Anaesth Unit/07

Emergency Drugs Chart 1



CALL for ASSISTANCE and CRASH CART

AIRWAY

1. CLEAR THE AIRWAY
2. INTUBATE
3. SECURE & INFLATE ET TUBE

CONTAIN ANY BLEEDING

BREATHING

1. CONNECT UP TO ANAESTHETIC MACHINE
2. VENTILATE with 100% OXYGEN @ 5-10 BPM ONLY

CARDIAC

Commence within 1 min of arrest

1. CHECK FOR HEART BEAT and/or PULSE
2. PRECORDIAL THUMP
3. EXTERNAL COMPRESSIONS @ 80-120/MIN

DRUGS

1. ESTABLISH IV ACCESS
2. HARTMANNS @ 10ml/kg/hr
3. ATROPINE*
4. ADRENALINE*

ECG

1. ASSESS RHYTHM

ASYSTOLE

Cardiac massage
*Adrenaline**

OR

FIBRILLATION

*Defibrillation***

CHECKLIST

BASIC LIFE SUPPORT:
Airway control
Ventilation with 100% O_2
Cardiac compressions
IV access
Fluids (type & rate)

DRUGS:
Atropine
Adrenaline
(repeat every 5 mins)
Reversing agents

MONITORING:
- *ECG*
- *Capnography (>15mmHg)*
- *Doppler (limb,tail or cornea)*
- *Pulse-oximetry*
- *Temp. probe*
- *PCV/TPP/glucose*
- *Blood gas analysis*

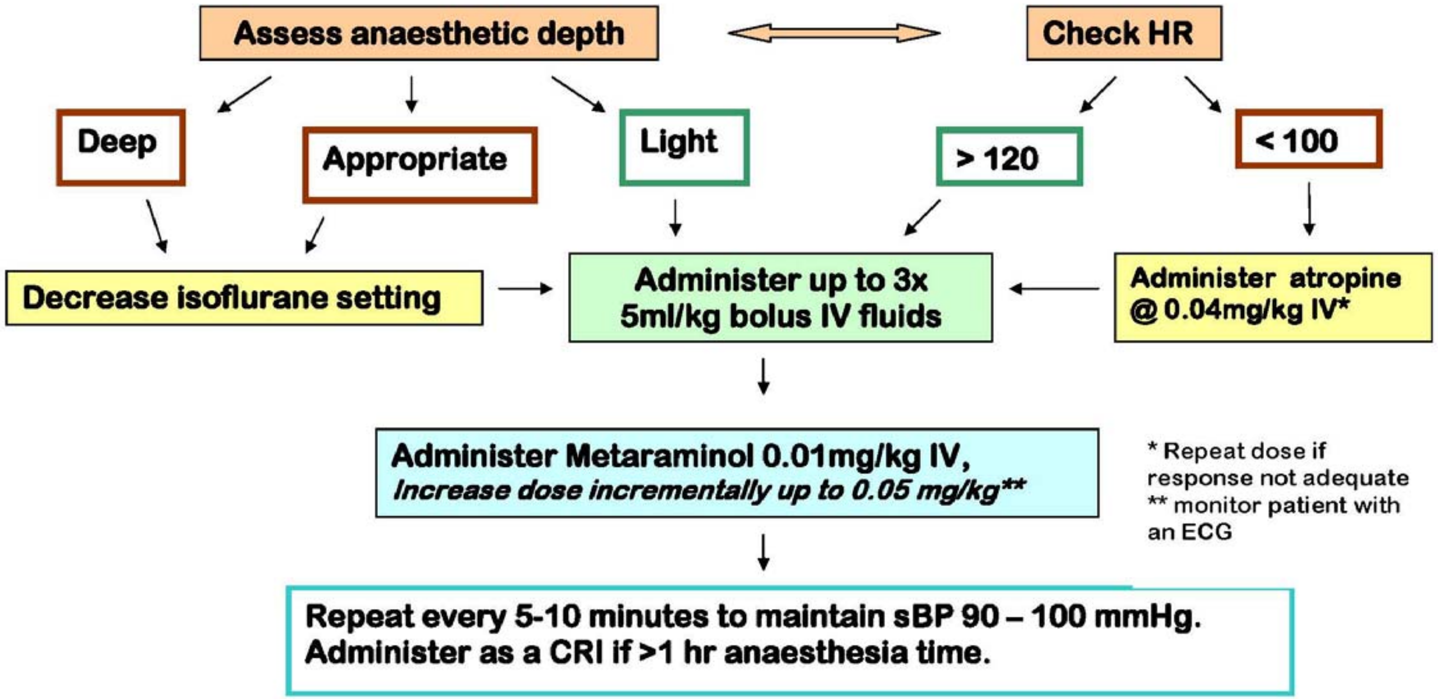
* refer to drug dose charts
** refer to defibrillator for instructions

UVCS/Anaesth Unit/07

KEEP A WRITTEN RECORD AT ALL TIMES

HYPOTENSION IN ASA 1 & 2 CATS with no Heart Disease

Systolic BP < 90mmHg



Assess anaesthetic depth

Deep

Appropriate

Light

Check HR

> 120

< 100

Decrease isoflurane setting

Administer up to 3x 5ml/kg bolus IV fluids

Administer atropine @ 0.04mg/kg IV*

Administer Metaraminol 0.01mg/kg IV, ***Increase dose incrementally up to 0.05 mg/kg****

* Repeat dose if response not adequate
** monitor patient with an ECG

Repeat every 5-10 minutes to maintain sBP 90 – 100 mmHg.
Administer as a CRI if >1 hr anaesthesia time.

Consider:
1. Reassessing anaesthetic depth regularly.
2. Reducing anaesthetic requirements.
- Administration of 66% N_2O if there are no contraindications
- Administration of analgesics (use opioids or LA techniques where indicated)

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NOTES:

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